

**Centers for Disease Control and Prevention
Healthcare Infection Control Practices Advisory Committee (HICPAC)**

**Draft Guideline for Environmental Infection Control in
Healthcare Facilities, 2001**

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Executive Summary

The *Guideline for Environmental Infection Control in Healthcare Facilities, 2001* is a compilation of recommendations for the prevention and control of infectious diseases that are linked to healthcare environments. This document: 1) updates and revises several sections (i.e., cleaning and disinfection of environmental surfaces, environmental sampling, laundry and bedding, and regulated medical waste) from the previous editions of the Centers for Disease Control and Prevention [CDC] document entitled *Guideline for Handwashing and Hospital Environmental Control*;^{1, 2} 2) incorporates discussions of air and water environmental issues from the *Guideline for the Prevention of Nosocomial Pneumonia*;³ 3) consolidates relevant environmental infection control measures from several other CDC guidelines;⁴⁻⁹ and 4) discusses two topics not addressed in previous CDC guidelines -- infection control issues related to the presence of animals in healthcare facilities, and water quality in hemodialysis settings.

Part I, “Background Information: Environmental Infection Control in Healthcare Facilities,” summarizes the major issues related to environmental infection control in healthcare facilities based on a comprehensive review of the scientific literature. Major attention is given to engineering and infection control concerns during construction, demolition, renovation, and repairs of healthcare facilities. Infection control measures used to recover from catastrophic events (e.g., flooding, sewage spills, loss of electricity and ventilation, disruption of the water supply) are reviewed. The limited impact of environmental surfaces, laundry, plants, animals, medical wastes, cloth furnishings, and carpeting on disease transmission in healthcare facilities is also explored.

Part II, “Recommendations for Environmental Infection Control in Healthcare Facilities,” presents control measures for preventing infections associated with air, water, or other environmental concerns within healthcare facilities as appropriate. These recommendations represent the consensus of the Healthcare Infection Control Practices Advisory Committee (HICPAC), a 12-member committee that advises CDC on issues related to the surveillance, prevention, and control of healthcare-associated infections primarily in United States healthcare facilities.¹⁰ As of January 1999, HICPAC expanded its infection control focus from acute-care hospitals to all venues where healthcare is provided (e.g., outpatient surgical centers, urgent care centers, clinics, outpatient dialysis centers, physicians’ offices, skilled nursing facilities). The topics addressed in this guideline are generally applicable to a variety of healthcare venues throughout the United States. This document is intended for use primarily by infection control practitioners, epidemiologists, employee health and safety personnel, engineers, informational system specialists, administrators, environmental service and housekeeping professionals, and architects for these facilities.

Whenever possible, the recommendations in Part II are based on data from well-designed scientific studies. Some studies, however, have been conducted in narrowly-defined patient populations or for specific healthcare settings (e.g., hospitals versus long-term care facilities), making generalization of their findings to all situations potentially problematic. Construction standards for hospitals or other healthcare facilities may not apply to residential home care units. Similarly, attempts to implement infection control measures indicated for immunosuppressed patient care are generally not necessary in those facilities where such patients are not present. Many of the recommendations are derived from empiric engineering concepts. Consequently, some of the recommendations may reflect an industry standard rather than an evidence-based conclusion. A few of the infection control measures proposed in this document cannot be rigorously evaluated for ethical or logistical reasons. Thus, some of the recommendations in Part II may be based on a strong theoretical rationale and suggestive evidence in the absence of confirmatory scientific evidence. Finally, some of the recommendations are derived from existing federal regulations. The references and the appendices comprise Parts III and IV, respectively.

This guideline also identifies key process measurement elements to assist facilities in monitoring compliance with the evidence-based Category IA or IB recommendations provided in Part II. These include: 1) conducting risk assessment prior to construction, renovation, demolition, or major repair projects; 2) conducting ventilation assessments related to construction barrier installation; 3) establishing and maintaining appropriate pressure differentials for special care areas [e.g., operating rooms, airborne infection isolation, protective environments]; 4) evaluating non-tuberculous mycobacteria culture results for possible environmental sources; and 5) implementing infection control procedures to prevent environmental spread of antibiotic-resistant gram-positive cocci and assuring compliance with these procedures.

This document does not discuss: 1) industrial hygiene concerns of a non-infectious nature [e.g., “sick building syndrome” from chemicals and fumes, allergies]; 2) environmental issues in the home; 3) home health care; 4) bioterrorism; and 5) foodborne illness acquired in healthcare facilities. This document includes only limited discussion of: 1) handwashing/hand hygiene; 2) Standard Precautions; 3) infection control measures used to prevent instrument or equipment contamination during patient care [e.g., preventing waterborne contamination of nebulizers or ventilator humidifiers]; and 4) infection control measures used to prevent exposures of patients and staff to potentially infectious substances. These topics are mentioned only if they are important in minimizing the transfer of pathogens to and from persons or equipment and the environment. Although the document discusses principles of cleaning and disinfection as they are applied to maintenance of environmental surfaces, the full discussion of sterilization and disinfection of medical instruments and direct patient-care devices is deferred to a future guideline. Similarly, the full discussion of handwashing/hand hygiene, which was a major section in the *Guideline for Handwashing and Hospital Environmental Control*, is deferred to a future guideline devoted to this single topic.

This guideline was prepared by CDC staff members from the National Center for Infectious Diseases (NCID) and the National Center for Chronic Disease Prevention and Health Promotion (NCCDPHP) and the designated HICPAC sponsor. Contributors to this document reviewed mostly English-language manuscripts identified from reference searches using the National Library of Medicine’s MEDLINE, bibliographies of published articles, and infection control textbooks. Working drafts of the guideline were reviewed by CDC scientists, HICPAC committee members, and experts in infection control, engineering, internal medicine, infectious diseases, epidemiology, and microbiology. All the recommendations may not reflect the opinions of all reviewers.

I. Background Information: Environmental Infection Control in Healthcare Facilities

A. Introduction

The healthcare environment contains a diverse population of microorganisms, but only a select few are significant pathogens for susceptible humans. Microorganisms are present in great numbers in moist, organic environments, but some can also persist under dry conditions. Although pathogenic microorganisms can be detected in air and water and on fomites, it is difficult to assess their role in causing infection and disease.¹¹ There are few reports which clearly delineate a “cause and effect” with respect to the environment, in particular for the housekeeping surfaces.

Seven levels of proof are used to evaluate the strength of evidence for an environmental source or means of transmission of infectious agents.¹¹ In the order of their rigor, these are: 1) the organism can survive after inoculation onto the fomite; 2) the organism can be cultured from in-use fomites; 3) the organism can proliferate in or on the fomite; 4) some measure of acquisition of infection cannot be explained by other recognized modes of transmission; 5) retrospective case-control studies show an association between exposure to the fomite and infection; 6) prospective observational studies may be possible when more than one similar type of fomite is in

use; and 7) prospective studies allocating exposure to the fomite to a subset of patients show an association between exposure and infection. An additional level of proof is that decontamination of the fomite results in the elimination of disease transmission.¹² Applying these proofs to disease investigations allows scientists to assess the contribution of the environment to disease transmission. The identification of a pathogen (e.g., vancomycin-resistant enterococci [VRE]) on an environmental surface during an outbreak serves as an illustration of this point. The presence of the pathogen does not automatically establish its causal role; its transmission from source to host could be through indirect means, such as via hand transferral.¹¹ The surface, therefore, would be considered one of a number of potential reservoirs for the pathogen, but not the “de facto” source of exposure.

An understanding of how infection occurs after exposure, based on the principles of the “Chain of Infection,” is also important in evaluating the contribution of the environment to healthcare-associated disease.¹³ All of the components of the “Chain” must be operational for infection to occur. That is, infection requires: 1) an adequate number of pathogenic organisms [dosage]; 2) pathogenic organisms of sufficient virulence; 3) a susceptible host; 4) an appropriate mode of transmission or transferral of the organism in sufficient number from a source to the host; and 5) the correct portal of entry into the host.

The presence of the susceptible host has focused recent attention on the importance of the healthcare environment and opportunistic pathogens in air and water and on fomites. As a result of advances in medical technology and therapies (e.g., intensification of cytotoxic chemotherapy; progress of transplantation medicine), a greater number of patients are becoming increasingly immunocompromised in the course of treatment and are therefore at increased risk of acquiring healthcare-associated opportunistic infections. Trends in healthcare delivery are also changing the distribution of patient populations and increasing the number of immunocompromised persons in healthcare settings other than acute-care hospitals, especially in light of early discharge of patients from care. According to the American Hospital Association (AHA), the number of hospitals in the United States in 1998 totaled 6,021, with 1,013,000 beds.¹⁴ This represents a 5.5% decrease in the number of acute-care facilities and a 10.2% decrease in the number of beds over the 5-year period 1994-1998.¹⁴ The total average daily census in U.S. acute-care hospitals in 1998 was 662,000 (65.4%) -- 36.5% less than the average daily census of 1,042,000 in 1978.¹⁴ As the number of acute-care hospitals declines, the length of stay in these facilities is concurrently decreasing, primarily for immunocompetent patients. Those patients remaining in acute-care facilities are likely to be those who require extensive medical interventions and are therefore at high risk for opportunistic infection.

The growing population of severely immunocompromised patients is at odds with demands on the healthcare industry to remain viable in the marketplace, to incorporate modern equipment, new diagnostic procedures, treatments, and to construct new facilities. Increasing numbers of healthcare facilities are likely to be faced with some construction in the near future as hospitals consolidate to reduce costs, defer care to ambulatory centers and satellite clinics, and try to create more “home-like” acute-care settings. In 1998, approximately 75% of the healthcare construction projects were for renovation or building outpatient facilities;¹⁵ the number of outpatient projects rose by 17% between 1998 and 1999.¹⁶ An aging population is also creating increasing demand for assisted-living facilities and skilled nursing centers. Construction of assisted-living facilities in 1998 rose by 49%, with 138 projects completed at a cost of \$703 million.¹⁶ Overall, from 1998 to 1999, healthcare construction costs increased by 28.5%, from \$11.56 billion to \$14.86 billion.¹⁶

Environmental disturbances associated with healthcare facility construction projects pose airborne and waterborne risks for the large number of patients who are at risk for healthcare-associated opportunistic infections. The increasing age of hospitals and healthcare facilities is also generating ongoing need for repair and remediation work (e.g., installing wiring for new information systems, removing old sinks, repairing elevator shafts) that can introduce or increase contamination of the air and water in patient-care environments. Aging

equipment, deferred maintenance, and natural disasters provide additional mechanisms for the entry of environmental pathogens into high-risk patient-care areas.

Architects, engineers, construction contractors, environmental health scientists, and industrial hygienists have historically directed the design and function of hospitals' physical plants. Increasingly, however, the growth in the number of susceptible patients and the increase in construction projects that can place these patients at risk for healthcare-associated infections call for the involvement of hospital epidemiologists and infection control professionals in plans for building, maintaining, and renovating healthcare facilities to minimize the adverse impact of the environment on the incidence of healthcare-associated infections. Examples of outbreaks which could have been prevented had this partnership been in place include: 1) transmission of infections due to *Mycobacterium tuberculosis*, varicella-zoster virus [VZV], and measles [rubeola] virus apparently facilitated by inappropriate air-handling systems in healthcare facilities;⁶ 2) disease outbreaks due to *Aspergillus* spp.,¹⁷⁻¹⁹ *Mucoraceae*,²⁰ and *Penicillium* spp. associated with the absence of environmental controls during periods of construction;²¹ 3) infections and/or colonizations of patients and staff with vancomycin-resistant *Enterococcus faecium* [VRE] and *Clostridium difficile*, presumably acquired in an indirect manner from contact with organisms present on environmental surfaces in healthcare facilities;²²⁻²⁵ and 4) outbreaks and pseudoepidemics of legionellae,^{26,27} *Pseudomonas aeruginosa*,²⁸⁻³⁰ and the nontuberculous mycobacteria [NTM]^{31,32} linked to water and aqueous solutions in healthcare facilities. The purpose of this guideline is to provide useful information for healthcare professionals and engineers alike in efforts to provide quality healthcare to their patients. The recommendations herein provide guidance to minimize and/or prevent transmission of pathogens in the indoor environment.

B. Key Terms Used in this Guideline

Although Appendix A provides definitions for terms discussed in Part I, several terms which pertain to specific patient-care areas and patients who are at risk for healthcare-associated opportunistic infection are presented here. Specific engineering parameters for these care areas are discussed more fully in the text. **Airborne Infection Isolation (AII)** refers to the isolation of patients infected with organisms that are spread via airborne droplet nuclei <5 µm in diameter. This isolation area is under negative pressure (i.e., externally exhausted), such that the direction of the air flow is from inside the room to the outdoors. The use of personal respiratory protection is also indicated for persons entering these rooms when occupied by a patient. A **Protective Environment (PE)** is a specialized patient-care area, usually in a hospital, with a positive air flow relative to the corridor (i.e., air flows from the room to the outside adjacent space). The combination of high efficiency particulate air (HEPA) filtration, high numbers of air changes per hour (ACH), and minimal leakage of air into the room creates an environment which can safely accommodate patients who have undergone allogeneic hematopoietic stem cell transplant (HSCT) and other patients with severe and prolonged neutropenia. **Immunocompromised patients** are those patients whose immune mechanisms are deficient because of immunologic disorders (e.g., human immunodeficiency virus [HIV] infection, congenital immune deficiency syndrome, chronic diseases [diabetes, cancer, emphysema, cardiac failure]) or immunosuppressive therapy (e.g., radiation, cytotoxic chemotherapy, anti-rejection medication, steroids). Immunocompromised patients who are identified as **high-risk patients** have the greatest risk of infection due to airborne or waterborne microorganisms. Patients in this subset include individuals who are severely neutropenic (i.e., <1,000 polymorphonuclear cells/µL for 2 weeks or <100 polymorphonuclear cells/mL for 1 week), allogeneic HSCT patients, and those who have received the most intensive chemotherapy (e.g., childhood amyeloid leukemia).

C. Air

1. Modes of Transmission of Airborne Diseases

A variety of airborne infections in susceptible hosts can result from exposures to clinically significant microorganisms released into the air when environmental reservoirs (i.e., soil, water, dust, and decaying organic

matter) are disturbed. Once these materials are brought indoors into a healthcare facility by any of a number of vehicles (e.g., people, air currents, water, construction materials, equipment), the attendant microorganisms can proliferate in a variety of indoor ecological niches and, if subsequently disbursed into the air, serve as a source for airborne healthcare-associated infections. Aerosolized oral and nasal secretions from patients represent another important source of pathogens that can be dispersed into the air.³³

Respiratory infections can be acquired from exposure to pathogens contained either in droplets or droplet nuclei. Exposure to microorganisms in droplets constitutes a form of direct contact transmission. When droplets are produced during a sneeze or cough, a cloud of infectious particles $>5\ \mu\text{m}$ in size is expelled, resulting in the potential exposure of susceptible persons within 3 feet of the source person.⁶ Examples of pathogens spread in this manner are influenza virus, rhinoviruses, adenoviruses, and respiratory syncytial virus (RSV). Since the transmission of these agents is largely direct and the droplets tend to fall out of the air quickly, measures to control air flow in a healthcare facility (e.g., use of negative pressure rooms) are not generally indicated for preventing the spread of diseases due to these agents. Strategies to control the spread of these diseases are outlined in another guideline.³

The spread of airborne infectious diseases via droplet nuclei is a form of indirect transmission.³⁴ Droplet nuclei are the residuals of droplets that, when suspended in air, subsequently dry and produce particles ranging in size from $1\ \mu\text{m}$ - $5\ \mu\text{m}$. These particles can: 1) contain potentially viable microorganisms; 2) be protected by a coat of dry secretions; 3) remain suspended indefinitely in air; and 4) be transported over long distances. The persistence of microorganisms in droplet nuclei is favored by dry, cool atmospheric conditions with little or no direct exposure to sunlight or other sources of radiation. Pathogenic microorganisms that can be spread via droplet nuclei include *M. tuberculosis*, VZV, and measles virus (rubeola).⁶ Several airborne pathogens have life-cycle forms that are similar in size to droplet nuclei and may exhibit similar behavior in the air. The spores of *Aspergillus fumigatus* have a diameter of $2\ \mu\text{m}$ - $3.5\ \mu\text{m}$, with a settling velocity estimated at 0.03 cm/sec, or about 1 meter/hour, in still air. With this enhanced buoyancy, the spores, which resist desiccation, can remain airborne indefinitely in air currents and travel far from their source.³⁵

2. Airborne Infectious Diseases in Healthcare Facilities

a. Aspergillosis and Other Fungal Diseases

Aspergillosis is caused by molds belonging to the genus *Aspergillus*. *Aspergillus* spp. are prototype healthcare-acquired pathogens associated with dusty or moist environmental conditions. Clinical and epidemiologic aspects of aspergillosis, summarized in Table 1, are discussed extensively in another guideline.³

Table 1. Clinical and Epidemiologic Characteristics of Aspergillosis

		References
Causative Agents	<i>Aspergillus fumigatus</i> (90% - 95% of <i>Aspergillus</i> infections among HSCT patients); <i>A. flavus</i> , <i>A. niger</i> , <i>A. terreus</i> , <i>A. nidulans</i> .	36 - 43
Mode of Transmission	Airborne transmission of fungal spores; direct inhalation; direct inoculation from environmental sources (rare)	37
Infection Associated With:	Construction, renovation, remodeling, repairs, building demolition; rare episodes associated with fomites	44 - 51
Clinical Syndromes and Diseases	Invasive disease is the most serious form of aspergillosis. Acute invasive: pneumonia; ulcerative tracheobronchitis; abscesses (aspergillomas) of the lungs, brain, liver, spleen, kidneys; thrombosis of deep blood vessels; osteomyelitis; necrotizing skin ulcers; endophthalmitis; sinusitis Chronic invasive: chronic pneumonitis Hypersensitivity: allergic bronchopulmonary aspergillosis Cutaneous: primary skin, burn wound infections	44, 45, 52 - 58
Patient Populations at Greatest Risk	HSCT patients; immunocompromised patients (i.e., those with underlying disease), patients undergoing chemotherapy, organ transplant recipients, preterm neonates, hemodialysis patients, patients with identifiable immune system deficiencies who receive care in general intensive care units, cystic fibrosis patients (may be colonized, occasionally become infected)	36, 59 - 78
Factors Influencing Severity and Outcome	Immune status of the patient, duration of severe neutropenia	79, 80
Occurrence	Rare, sporadic but increasing as proportion of immunocompromised patients increases; 5% of HSCT patients infected, <5% in solid organ transplant recipients	36, 37, 81 - 88
Mortality Rate	≤100% if severe neutropenia persists; 13% - 80% in leukemia patients	58, 83, 89, 90
Antifungal Therapy	Yes - amphotericin B (AmB, liposomal AmB), itraconazole; mortality due to invasive aspergillosis remains high among severely neutropenic patients despite treatment	37

Aspergillus spp. are ubiquitous aerobic fungi that occur in soil, water, and decaying vegetation; the organism also survives well in air, dust, and moisture present in healthcare facilities.^{91 - 93} The presence of aspergilli in the healthcare facility environment is the most important extrinsic risk factor for opportunistic invasive aspergillosis.^{69, 94} Site renovation and construction can disturb *Aspergillus*-contaminated dust and produce bursts of airborne fungal spores. Increased levels of atmospheric dust and fungal spores have been associated with clusters of healthcare-acquired infections in immunocompromised patients.^{17, 20, 44, 47, 49, 50, 95 - 98} Absorbent building materials (e.g., wallboard) serve as an ideal substrate for the proliferation of this organism if they become and remain wet, thereby increasing the numbers of fungal spores in the area. Patient-care items, devices, and equipment can become contaminated with *Aspergillus* spp. spores and serve as sources of infection if stored in such areas.⁵⁷

Most cases of aspergillosis are caused by *Aspergillus fumigatus*, a thermotolerant/thermophilic fungus capable of growing over a temperature range from 12°C - 53°C (53.6°F - 127.4°F); optimal growth occurs at approximately 40°C (104°F), a temperature inhibitory to most other saprophytic fungi.⁹⁹ It can use cellulose or sugars as carbon sources; because its respiratory process requires an ample supply of carbon, decomposing organic matter is an ideal substrate.

Other opportunistic fungi that have been occasionally linked with healthcare-associated infections are members of the order *Mucorales* (e.g., *Rhizopus* spp.) and miscellaneous moniliaceous molds (e.g., *Fusarium* spp., *Penicillium* spp.). Many of these fungi can proliferate in moist environments, such as water-damaged wood and building materials. Some fungi (e.g., *Fusarium* spp., *Pseudoallescheria* spp.) can be airborne pathogens as well.¹⁰⁰ Some of these agents and

their sources in the healthcare environment are listed in Table 2. As with aspergillosis, a major risk factor for disease caused by any of these pathogens is the host's severe immunosuppression from either underlying disease or immunosuppressive therapy.^{101, 102}

Table 2. Environmental Fungal Pathogens and Their Entry into the Healthcare Facility

Implicated Environmental Vehicle	References
<i>Aspergillus</i> spp.	
Improperly functioning ventilation systems	20, 46, 47, 97, 98, 103, 104
Air filters ^{a, b}	17, 18, 105, 106, 107
Frames of air filters	17, 18
Window air conditioners	96
Backflow of contaminated air	107
Air exhaust contamination ^b	104
False ceilings	48, 57, 97, 108
Fibrous insulation and perforated metal ceiling	66
Acoustic ceiling tiles, plasterboard	18, 109
Fireproofing material	48, 49
Damp wood building materials	49
Opening doors to construction site	110
Construction	69
Open windows	20, 108, 111
Disposal conduit door	68
Hospital vacuum cleaner	68
Elevator	112
Arm boards	57
Walls	113
Unit Kitchen	114
Food	21
Ornamental plants	21
<i>Mucorales / Rhizopus</i> spp.	
Air filter	20, 115
False ceilings	97
Heliport	115
<i>Scedosporium</i> spp.	
Construction	116
<i>Penicillium</i> spp.	
Rotting cabinet wood, pipe leak	21
Ventilation duct fiberglass insulation	112
Air filters	105
Topical anesthetic	117
<i>Acremonium</i> spp.	
Air filters	105
<i>Cladosporium</i> spp.	
Air filters	105
<i>Sporothrix</i>	
Construction (pseudoepidemic)	118

a. Pigeons, their droppings and roosts are associated with spread of *Aspergillus*, *Cryptococcus*, and *Histoplasma* spp. There have been at least three outbreaks linked to contamination of the filtering systems from bird droppings.^{97, 103, 104} Pigeon mites may gain access into a health care facility through the ventilation system.¹¹⁹

b. American Institute of Architects [AIA] standards stipulate that: 1) exhaust outlets are to be placed >25 feet from air intake systems; 2) the bottom of outdoor air intakes for HVAC systems be 6 feet above ground or 3 feet above roof level; and 3) exhaust outlets from contaminated areas are situated above the roof level and arranged to minimize the recirculation of exhausted air back into the building.¹²⁰

Infections due to *Cryptococcus neoformans*, *Histoplasma capsulatum*, or *Coccidioides immitis* can potentially occur in healthcare settings if nearby ground is disturbed and a malfunction of the facility's air-intake components allows these pathogens to enter the ventilation system. *C. neoformans* is a yeast <2µm in diameter found in soil contaminated with bird droppings, particularly from pigeons.^{98, 103, 104, 121} *H. capsulatum*, with particle diameters ranging from 2µm - 5 µm, is endemic in the soil of the central river valleys of the United States. Large numbers of these infectious particles are found associated with chicken coops and the roosts of blackbirds.^{98, 103, 104, 122} Several outbreaks of histoplasmosis have been associated with disruption of the environment; construction activities in an endemic area may be a potential risk factor for healthcare-acquired airborne infection.^{123, 124} *C. immitis*, with an infectious particle of 3µm - 5 µm diameter, has similar potential, especially in the endemic southwestern United States and during seasons of drought followed by heavy rainfall. After the 1994 earthquake centered near Northridge, California, the incidence of coccidioidomycosis in the surrounding area exceeded the historical norm.¹²⁵

Emerging evidence suggests that *Pneumocystis carinii*, now classified as a fungus, may be spread via airborne person-to-person transmission.¹²⁶ Controlled studies in animals first demonstrated that *P. carinii* could be spread through the air.¹²⁷ More recent studies in healthcare settings have detected nucleic acids of *P. carinii* in air samples from areas frequented or occupied by *P. carinii*-infected patients but not in control areas with no infected patients.^{128, 129} Despite the earlier assumption that *P. carinii* pneumonia (PCP) was not spread from person-to-person, clusters of cases have been identified among immunocompromised patients who had contact with a source patient and with each other. Recent studies have examined the presence of *P. carinii* DNA in oropharyngeal washings and the nares of infected patients, their direct contacts, and persons with no direct contact.^{130, 131} Molecular analysis of the DNA by polymerase chain reaction (PCR) supports air spread of *P. carinii* from infected patients to direct contacts, but immunocompetent contacts tend to become transiently colonized rather than infected.¹³¹ The role of colonized persons in the spread of PCP remains to be determined. At present, specific modifications to ventilation systems to control spread of PCP in a healthcare facility are not indicated. Current recommendations outline isolation procedures to minimize or eliminate contact of immunocompromised patients not on PCP prophylaxis with PCP-infected patients.^{6, 132}

b. Tuberculosis and Other Bacterial Diseases

The prototype bacterium associated with airborne transmission is *Mycobacterium tuberculosis*. A comprehensive review of the microbiology and epidemiology of *M. tuberculosis* and guidelines for tuberculosis (TB) infection control have been published.^{4, 133, 134} Table 3 summarizes clinical and epidemiologic information from these materials. *M. tuberculosis* is carried by droplet nuclei generated when persons, primarily adults and adolescents, who have pulmonary or laryngeal TB sneeze, cough, speak, or sing;¹³⁵ normal air currents can keep these particles airborne for prolonged periods and spread them throughout a room or building.¹³⁶

Table 3. Clinical and Epidemiologic Characteristics of Tuberculosis^{4, 133 - 135, 137 - 140}

Causative Agents	<i>Mycobacterium tuberculosis</i> , <i>M. bovis</i> , <i>M. africanum</i>
Mode of Transmission	Airborne transmission via droplet nuclei 1 µm - 5 µm in diameter
Patient Factors Associated with Infectivity and Transmission	Disease of the lungs, airways, or larynx; presence of cough or other forceful expiratory measures; presence of acid-fast bacilli (AFB) in the sputum; failure of the patient to cover the mouth and nose when coughing or sneezing; presence of cavitation on chest radiograph; inappropriate or shortened duration of chemotherapy
Infections Associated With:	Exposures in relatively small, enclosed spaces; inadequate ventilation resulting in insufficient removal of droplet nuclei; cough-producing procedures performed in areas without proper environmental controls; recirculation of air containing infectious droplet nuclei
Clinical Syndromes & Diseases	Pulmonary TB ; extrapulmonary TB can affect any organ system or tissue; laryngeal TB is highly contagious
Populations at Greatest Risk	Immunocompromised persons (e.g., HIV-infected persons), medically-underserved persons, urban poor, substance abusers, homeless persons, elderly persons, migrant farm workers, present and former prison inmates, close contacts of known cases, foreign-born persons from areas with high prevalence of TB, healthcare workers
Factors Influencing Severity and Outcome	Concentration of droplet nuclei in air, duration of exposure; age at infection; immunosuppression due to therapy or disease; underlying chronic medical conditions, history of malignancies or lesions of the lungs
Occurrence	Worldwide; incidence in the U.S. is approximately 8 cases /100,000 population (1997). ¹⁴⁰
Mortality	1,202 deaths in the U.S. (1996). ¹⁴⁰
Chemoprophylaxis / Treatment	Preventive therapy post infection: isoniazid (INH) or rifampin (RIF) and pyrazinamide (PZA) as indicated ^{4, 134, 137, 139} Directly observed therapy [DOT] for active cases: INH, RIF, PZA, ethambutol (EMB), streptomycin (SM) in combinations determined by prevalent levels of specific resistance. ^{4, 134, 137, 139} Consult therapy guidelines for specific treatment indications.

Gram-positive cocci (i.e., *Staphylococcus aureus*, group A beta-hemolytic streptococci), also important healthcare-associated pathogens, are resistant to inactivation by drying and can persist in the environment and on environmental surfaces for extended periods. These organisms can be shed from heavily colonized persons and discharged into the air. Airborne dispersal of *S. aureus* is directly related to the concentration of the bacterium in the anterior nares.¹⁴¹ Approximately 10% of healthy carriers will disseminate *S. aureus* into the air, and some persons become more effective disseminators of *S. aureus* than others.^{142 - 146} The dispersal of *S. aureus* into air can be exacerbated by concurrent viral upper respiratory infection, thereby turning a carrier into a “cloud shedder.”¹⁴⁷ Outbreaks of surgical site infections (SSIs) caused by group A beta-hemolytic streptococci have been traced to airborne transmission from colonized operating room personnel to patients.^{148 - 151} In these situations, the strain causing the outbreak was recovered from the air in the operating room^{148, 149, 152} or on settle plates in a room in which the carrier exercised.^{149 - 151} *S. aureus* and group A streptococci have not been linked to airborne transmission outside of operating rooms, burn units, and neonatal nurseries.^{153, 154} Transmission of these agents also occurs via contact (*S. aureus*) and droplets (group A beta-hemolytic streptococci).

Other gram-positive bacteria linked to airborne transmission include *Bacillus* spp. which are capable of sporulation as environmental conditions become less favorable to support their growth. Outbreaks and pseudo-outbreaks have been attributed to *B. cereus* in maternity, pediatric, intensive care, and bronchoscopy units; many of these episodes were secondary to environmental contamination.^{155 - 158}

Gram-negative bacteria are rarely associated with episodes of airborne transmission because they generally require moist environments for persistence and growth. The main exception is *Acinetobacter* spp. which can withstand the inactivating effects of drying. In one epidemiologic investigation of bloodstream infections among pediatric patients, identical *Acinetobacter* spp. were cultured from the patients, air, and room air conditioners in a nursery.¹⁵⁹ Aerosols generated from showers and faucets may potentially contain legionellae and other gram-negative waterborne bacteria (e.g., *Pseudomonas aeruginosa*). Exposure to these organisms is through direct inhalation. However, since water is the source of the organisms and exposure occurs in the vicinity of the aerosol, the discussion of the diseases associated with such aerosols and the prevention measures used to curtail their spread is deferred to the Water portion of Part I.

c. Airborne Viral Diseases

Some human viruses are transmitted from person to person via droplet aerosols, but very few viruses are consistently airborne in transmission (i.e., routinely suspended in an infective state in air and capable of spreading great distances), and healthcare-associated outbreaks of airborne viral disease are limited to a few agents. Consequently, infection control measures used to prevent spread of these viral diseases in healthcare facilities primarily involve patient isolation, vaccination of susceptible persons, and antiviral therapy as appropriate rather than measures to control air flow or quality.⁶ Infections due to VZV are frequently described in healthcare facilities. Healthcare-associated airborne outbreaks of VZV infections from patients with primary infection and disseminated zoster have been documented; patients with localized zoster have, on rare occasions, also served as source patients for outbreaks in healthcare facilities.^{160 - 164} VZV infection can be prevented by vaccination, although patients who develop a rash within 6 weeks of receiving varicella vaccine or who develop breakthrough varicella following exposure should be considered contagious.¹⁶⁵

In a limited number of instances, viruses whose major mode of transmission is via droplet contact have been shown to cause clusters of infections in group settings through airborne routes. The factors facilitating airborne distribution of these viruses in an infective state are unknown, but a presumed requirement is a source patient in the early stage of infection who is shedding large numbers of viral particles into the air. Airborne transmission of measles has been documented in healthcare facilities.^{166 - 169} Institutional outbreaks of influenza virus infections have occurred predominantly in nursing homes,^{170 - 174} and less frequently in medical and neonatal intensive care units, chronic care areas, HSCT units, and pediatric wards.^{175 - 178} There is some evidence supporting airborne transmission of influenza viruses by droplet nuclei,^{179 - 180} and case clusters in pediatric wards suggest that droplet nuclei may play a role in transmitting respiratory pathogens such as adenoviruses and RSV.^{175, 181, 182} Some suggestive evidence also supports airborne transmission of enteric viruses. A large outbreak of a Norwalk-like virus infection involving more than 600 staff personnel over a 3 week period was investigated in a Toronto, Ontario hospital in 1985. Common sources such as food or water were ruled out during the investigation, leaving airborne spread as the most likely candidate for transmission.¹⁸³

Smallpox virus, a potential agent of bioterrorism, is spread predominantly via direct contact with infectious droplets, but can be associated with airborne transmission.^{184, 185} A German hospital study from 1970 documented the ability of this virus to spread over considerable distances and cause infection at low doses in a well-vaccinated population; factors potentially facilitating transmission in this situation included a patient with cough and an extensive rash, indoor air with low relative humidity, and faulty ventilation patterns due to hospital design.¹⁸⁶ Smallpox patients with extensive rash are more likely to have lesions present on mucous membranes and therefore have greater potential to disseminate virus into the air.¹⁸⁶ Two cases of laboratory-acquired smallpox virus infection in the United Kingdom in 1978 were also thought to be due to airborne transmission.¹⁸⁷

Airborne transmission may play a role in the natural spread of hantaviruses and certain hemorrhagic fever viruses (e.g., Ebola, Marburg, Lassa), but evidence for airborne spread of these agents in healthcare facilities is inconclusive.¹⁸⁸ Although hantaviruses can be transmitted when aerosolized from rodent excreta,^{189, 190} person-to-person spread of hantavirus infection from source patients has not occurred in healthcare facilities.^{191 - 193} Nevertheless, healthcare workers are advised to contain potentially infectious aerosols and wear NIOSH-approved respiratory protection when working with this agent in laboratories or autopsy suites.¹⁹⁴ Lassa virus transmission via aerosols has been demonstrated in the laboratory and incriminated in healthcare-associated infections in Africa,^{195 - 197} but airborne spread of this agent in hospitals of developed nations appears to be inefficient.^{198, 199} Viral hemorrhagic diseases primarily

occur after direct exposure to infected blood and body fluids, and the use of standard and droplet precautions is sufficient to prevent transmission early in the course of these illnesses.²⁰⁰ However, it is unclear whether these viruses can persist in droplet nuclei that might remain after droplet production from coughs or vomiting in the latter stages of illness.²⁰¹ Although the use of a negative-pressure room is not required during the early stages of illness, its use might be prudent at the time of hospitalization to avoid the need for subsequent patient transfer. CDC guidelines recommend negative-pressure rooms with anterooms for patients with hemorrhagic fever and use of HEPA respirators by persons entering these rooms when the patient has prominent cough, vomiting, diarrhea, or hemorrhage.^{6, 200}

Table 4. Microorganisms Associated with Airborne Transmission^a

	Fungi	Bacteria	Viruses
Numerous Reports in Health Care	<i>Aspergillus</i> spp. ^b <i>Mucorales (Rhizopus</i> spp.) ^{97, 115}	<i>Mycobacterium tuberculosis</i> ^b	Measles (rubeola) virus ^{166 - 168} Varicella-zoster virus ^{160 - 164}
Atypical, Occasional Reports	<i>Acremonium</i> spp. ^{105, 202} <i>Fusarium</i> spp. ¹⁰² <i>Pseudoallescheria boydii</i> ¹⁰⁰ <i>Scedosporium</i> spp. ¹¹⁶ <i>Sporothrix cyanescens</i> ^{d 118}	<i>Acinetobacter</i> spp. ¹⁵⁹ <i>Bacillus</i> spp. ^{d 158, 203} <i>Brucella</i> spp. ^{e 204 - 207} <i>Staphylococcus aureus</i> ^{146, 154} Group A <i>Streptococcus</i> ¹⁴⁹	Smallpox virus (variola) ^{c 186, 187} Influenza viruses ^{179, 180} Respiratory syncytial virus ¹⁸¹ Adenoviruses ¹⁸² Norwalk-like virus ¹⁸³
Airborne in Nature; No Airborne Cases in Health Care	<i>Coccidioides immitis</i> ¹²⁵ <i>Cryptococcus</i> spp. ¹²¹ <i>Histoplasma capsulatum</i> ¹²⁴	<i>Coxiella burnetii</i> (Q fever) ²⁰⁸	Hantaviruses ^{191, 193} Lassa virus ²⁰¹ Marburg virus ²⁰¹ Ebola virus ²⁰¹ Crimean-Congo virus ²⁰¹
Under Investigation	<i>Pneumocystis carinii</i> ¹³¹	-	-

a. This list excludes microorganisms transmitted from aerosols derived from water.

b. Refer to the text for references for these diseases.

c. Airborne transmission of smallpox is controversial. Potential for airborne transmission increases with patients who are effective disseminators present in facilities with low relative humidity in the air and faulty ventilation.

d. Documentation of pseudoepidemic during construction.

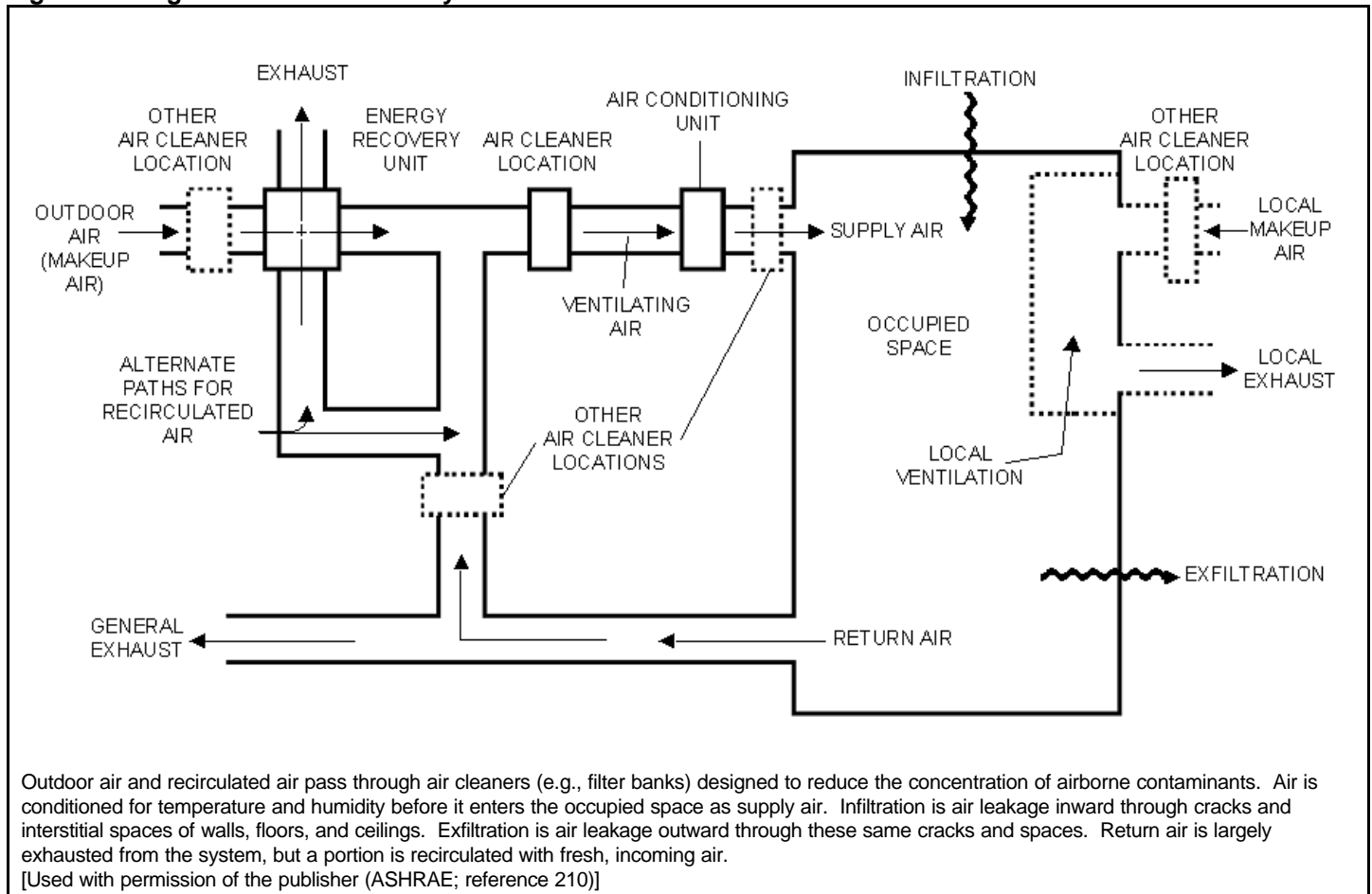
e. Airborne transmission in the laboratory only.

3. Heating, Ventilation, and Air Conditioning Systems in Healthcare Facilities

a. Basic Components and Operations

Heating, ventilation, and air conditioning (HVAC) systems in healthcare facilities are designed to: 1) maintain the indoor air temperature and humidity at comfortable levels for staff, patients, and visitors; 2) control odors; 3) remove contaminated air; 4) facilitate air-handling requirements to protect susceptible staff and patients from airborne healthcare-associated pathogens; and 5) minimize the risk of transmission of airborne pathogens from infected patients.^{35, 120} An HVAC system includes an air inlet or intake; filters; humidity modification mechanisms (i.e., humidity control in summer, humidification in winter); heating and cooling equipment; fans; ducts; air exhaust or outtakes; and registers, diffusers, or grilles for proper distribution of the air (Figure 1).^{209, 210} Decreased performance of healthcare facility HVAC systems, filter inefficiencies, improper installation, and poor maintenance can contribute to the spread of healthcare-associated airborne infections.

Figure 1. Diagram of a Ventilation System^a



The American Institute of Architects (AIA) has published guidelines for the design, construction, and renovation of healthcare facilities that include indoor air-quality standards (e.g., ventilation rates, temperature levels, humidity levels, pressure relationships, minimum air changes per hour [ACH]) specific to each zone or area in healthcare facilities (e.g., operating rooms, laboratories, diagnostic areas, patient-care areas, support departments).¹²⁰ These guidelines represent a consensus document among authorities having jurisdiction (AHJ), governmental regulatory agencies (i.e., Department of Health and Human Services [DHHS]; Department of Labor, Occupational Safety and Health Administration [OSHA]), healthcare professionals, professional organizations (e.g., American Society of Heating, Refrigeration, and Air-conditioning Engineers [ASHRAE], American Society of Healthcare Engineers [ASHE]), and accrediting organizations (i.e., Joint Commission on Accreditation of Healthcare Organizations [JCAHO]). Many state or local agencies that license healthcare facilities have either incorporated or adopted by reference these guidelines into their state standards. The JCAHO, through its surveys, assures that facilities are in compliance with the space and square footage requirements of this standard for new construction.

Recommendations for engineering controls to contain or prevent the spread of airborne contaminants center on: 1) local exhaust ventilation [i.e., source control]; 2) general ventilation; and 3) air cleaning.⁴ General ventilation encompasses: 1) dilution and removal of contaminants via filtration and air changes per hour [ACH]; 2) airflow patterns in rooms or areas; 3) airflow direction in facilities; and 4) pressure differentials for special-care areas.

A centralized HVAC system operates as follows. Outdoor air enters the system, where low- efficiency or “roughing” filters remove large particulate matter and many microorganisms. The air enters the distribution system for conditioning to appropriate temperature and humidity levels, passes through an additional bank of filters for further cleaning, and is delivered to each zone of the building. After the conditioned air is distributed to the designated space, it is withdrawn through a return duct system and delivered back to the HVAC unit. A portion of this “return air” is exhausted to the outside while the remainder is mixed with outdoor air and filtered for dilution and removal of contaminants.²¹¹ Air

from toilet rooms or other soiled areas is usually exhausted directly to the atmosphere through a separate duct exhaust system. Air from rooms housing tuberculosis patients is exhausted to the outside if possible, or passed through a HEPA filter before recirculation. UVGI can be used as an adjunct air-cleaning measure but cannot replace HEPA filtration.

b. Filtration

i. Filter Types and Methods of Filtration

Filtration, the physical removal of particulates from air, is the first step in achieving acceptable indoor air quality. Filtration is the primary means of cleaning the air. There are five methods of filtration (Table 5). During filtration, outdoor air passes through two filter beds or banks, with efficiencies of 20% - 40% and the second $\geq 90\%$, respectively, for a combined efficiency of nearly 100% in removing particles 1 μm - 5 μm in diameter.³⁵ The low-to-medium efficiency filters in the first bank have low resistance to airflow, but this feature tends to allow some small particulates to pass onto heating and air conditioning coils and into the indoor environment.³⁵ Incoming air is mixed with recirculated air and reconditioned for temperature and humidity before being filtered by the second bank of filters. The performance of filters with $\leq 90\%$ efficiency is measured using either the dust spot test or the weight-arrestance test.³⁵

Table 5. Filtration Methods²¹²

Basic Method	Principle of Performance	Filtering Efficiency
Straining	Particles in the air are larger than the openings between the filter fibers. Gross removal of large particles.	Low
Impingement	Particles collide with filter fibers and remain attached to the filter. Fibers may be coated with adhesive.	Low
Interception	Particles enter into the filter and become entrapped and attached to the filter fibers.	Medium
Diffusion	Small particles, moving in erratic motion, collide with filter fibers and remain attached.	High
Electrostatic	Particles bearing negative electrostatic charge are attracted to filter with positively-charged fibers.	High

The second filter bank usually consists of high-efficiency filters. High-efficiency air filtration systems can provide air that is almost particle free. This filtration system is adequate for most patient-care areas in ambulatory care facilities and hospitals, including the operating room environment and areas providing central services.¹²⁰ Nursing facilities may use 90% dust-spot efficient filters as the second bank of filters,¹²⁰ while a HEPA filter bank may be indicated for special-care areas of hospitals. HEPA filters are at least 99.97% efficient for removing particles $\geq 0.3 \mu\text{m}$ in diameter. (As a reference, *Aspergillus* spores are 2.5 - 3 μm in diameter.) Examples of care areas where HEPA filters are used include rooms housing severely neutropenic patients and those operating rooms designated for orthopedic implant procedures.³⁵

Maintenance costs associated with HEPA filters are high when compared to other types of filters, but use of in-line disposable prefilters can increase the life of a HEPA filter by approximately 25%. Alternatively, if a disposable prefilter is followed by a 90%-efficient filter, the life of the HEPA filter can be extended up to 900%. This concept, called “progressive filtration,” allows HEPA filters in special care areas to be used for 10 years or more.²⁰⁹ HEPA filter efficiency is monitored with the dioctylphthalate (DOP) particle test using particles that are 0.3 μm in diameter.

HEPA filters are usually framed with metal, although some older versions have wood frames. A metal frame has no advantage over a properly fitted wood frame with respect to performance, but wood can compromise the air quality if it becomes and remains wet, allowing the growth of fungi and bacteria. Hospitals are therefore advised to phase out water-damaged or spent wood-framed filter units and replace these with metal-framed HEPA filters.

HEPA filters are usually fixed into the HVAC system, but portable, industrial grade HEPA units are available which filter air at the rate of 300-800 ft³/min are also available. Portable HEPA filter are used to: 1) temporarily recirculate air in rooms with no general ventilation; 2) augment systems that cannot provide adequate airflow; or 3) provide increased effectiveness in airflow.⁴ Portable HEPA units are useful engineering controls when the central HVAC system is undergoing repairs,²¹³ but these units do not satisfy fresh air requirements.²¹⁰ The effectiveness of the portable unit for particle removal is dependent on: 1) the configuration of the room; 2) the furniture and persons in the room; 3) the placement of the units relative to the contents and layout of the room; and 4) the location of the supply and exhaust registers or grilles. If portable, industrial-grade units are used, they should be capable of recirculating all or nearly all of the room air through the HEPA filter, and the unit should be designed to achieve the equivalent of ≥ 12 air changes per hour (ACH).⁴ (An average room has approximately 1600 ft³ of airspace).

ii. Filter Maintenance

Efficiency of the filtration system is dependent on the density of the filters that may create a pressure drop unless compensated by stronger and more efficient fans so that flow of air is maintained. For optimal performance, filters require monitoring and replacement in accordance with the manufacturer's recommendations and standard preventive maintenance practices.²¹⁴ Excess accumulation of dust and particulates increases filter efficiency, requiring more pressure to push the air through. The pressure differential across filters is measured by use of manometers or other gauges. A pressure reading that exceeds specifications indicates the need to change the filter. Filters also require regular inspection for other potential causes of decreased performance. Gaps in and around filter banks and heavy soil and debris upstream of poorly-maintained filters have been implicated in healthcare-associated outbreaks of aspergillosis, especially during times of nearby construction.^{17, 18, 106, 215}

c. Ultraviolet Germicidal Irradiation (UVGI)

As a supplemental air-cleaning measure, UVGI is effective in reducing the transmission of airborne bacterial and viral infections in hospitals, military housing, and classrooms, but it has only a minimal inactivating effect on fungal spores.²¹⁶ -²²¹ UVGI is also used in air handling units to prevent or limit the growth of vegetative bacteria and fungi. Most commercially available UV lamps used for germicidal purposes are low-pressure mercury vapor lamps that emit radiant energy predominantly at a wave-length of 253.7 nm.^{222, 223} Two systems of UVGI have been used in healthcare settings - duct irradiation and upper-room air irradiation. In duct irradiation systems, UV lamps are placed inside ducts that remove air from rooms to disinfect the air before it is recirculated. When properly designed, installed, and maintained, high levels of UVGI can be attained in the ducts with little or no exposure of persons in the rooms.^{224, 225} In upper-room air irradiation, UV lamps are either suspended from the ceiling or mounted on the wall.⁴ Upper air UVGI units have two basic designs: 1) a "pan" fixture with UVGI unshielded above the unit so to direct the irradiation upward; or 2) a fixture with a series of parallel plates to columize the irradiation outward while preventing the light from getting to the eyes of the room's occupants. The germicidal effect is dependent on air mixing via convection between the room's irradiated upper zone and the lower patient-care zones.^{226, 227}

Bacterial inactivation studies using BCG mycobacteria and *Serratia marcescens* have estimated the effect of UVGI as equivalent to 10 ACH - 39 ACH.^{228, 229} Another study, however, suggests that UVGI may result in fewer equivalent ACH in the patient-care zone, especially if the mixing of air between zones is insufficient.²²⁷ The use of fans or HVAC systems to generate air movement may increase the effectiveness of UVGI if airborne microorganisms are exposed to the light energy for a sufficient length of time.^{226, 228, 230 - 232} The optimal relationship between ventilation and UVGI is not known.

Because the clinical effectiveness of UV systems may vary, UVGI is not recommended for air management prior to air recirculation from airborne isolation rooms. It is also not recommended as a substitute for HEPA filtration, local exhaust of air to the outside, or negative pressure.⁴ The use of UV lamps and HEPA filtration in a single unit offers little or no infection control benefits over those provided by the use of a HEPA filter alone.²³³ Duct systems with UVGI are not recommended as a substitute for HEPA filters if the air from isolation rooms must be recirculated to other areas of the facility.⁴ Regular maintenance of UVGI systems is crucial and usually consists of keeping the bulbs free of dust and replacing old bulbs as necessary. Safety issues associated with the use of UVGI systems are described in other guidelines.⁴

d. Conditioned Air in Occupied Spaces

Two essential components of conditioned air are temperature and humidity. After outside air passes through a low- or medium-efficiency filter, the air undergoes conditioning for temperature and humidity control before it passes through high-efficiency or HEPA filtration.

i. Temperature

HVAC systems in healthcare facilities have either single-duct or dual-duct systems.^{35, 234} A single-duct system distributes cooled air (12.8°C [55°F]) throughout the building, and uses thermostatically controlled reheat boxes located in the terminal ductwork to warm the air for individual or multiple rooms. The more common dual-duct system consists of parallel ducts, one with a cold air stream and the other providing a hot air stream. A mixing box in each room or group of rooms mixes the two air streams to achieve the desired temperature. Temperature standards are given as either a single temperature or a range, depending on the specific healthcare zone. Cool temperature standards (20°C - 23°C [68°F - 73°F]) are usually associated with operating rooms, clean workrooms, and endoscopy suites.¹²⁰ A warmer temperature (24°C [75°F]) is needed in areas requiring greater degrees of patient comfort. Most other zones use a temperature range of 21°C - 24°C (70°F - 75°F).¹²⁰ Temperatures outside of these ranges may be needed on limited occasions in limited areas depending on individual circumstances during patient care (e.g., cooler temperatures in operating rooms during specialized operations).

ii. Humidity

Four measures of humidity are used to quantify different physical properties of the mixture of water vapor and air. The most common of these is “relative humidity,” which is the ratio of the amount of water vapor in the air to the amount of water vapor air can hold at that temperature.²³⁵ The other measures of humidity are specific humidity, dew point, and vapor pressure.²³⁵

Relative humidity measures the percentage of saturation. At 100% relative humidity, the air is saturated. For most areas within healthcare facilities, the designated comfort range is 30% - 60% relative humidity.^{120, 210} Relative humidity levels >60%, in addition to being perceived as uncomfortable, promote fungal growth.²³⁶ Humidity levels can be manipulated by either of two mechanisms.²³⁷ In a water-wash unit, water is sprayed and drops are taken up by the filtered air; additional heating or cooling of this air sets the humidity levels. The second mechanism is by means of water vapor created from steam and added to filtered air in humidifying boxes.

iii. Ventilation

The control of air pollutants (e.g., microorganisms, dust, chemicals, smoke) at the source is the most effective way to maintain clean air. The second most effective means of controlling indoor air pollution is through ventilation. Ventilation supply rates are historically based on the need to control odors and carbon dioxide levels.²³⁸ Ventilation rates are voluntary unless a state or local government specifies a standard in healthcare licensing or health department requirements. These standards typically apply to only the design of a facility, rather than its operation.^{214, 239} Based on the scientific knowledge and professional judgment reflected in the AIA guidelines, ASHRAE has developed ventilation standards designed primarily to satisfy the odor criterion.²³⁸ ASHRAE and the American National Standards Institute (ANSI) have produced design recommendations for ventilation and pressure relationships for various patient-care areas.²¹⁰ Healthcare facilities without specific ventilation standards should follow ANSI/ASHRAE Standard 62, *Ventilation for Acceptable Indoor Air Quality*.^{210, 234}

Ventilation guidelines are defined in terms of air volume per minute per occupant, and are based on the assumption that occupants and their activities are responsible for most of the contaminants in the conditioned space.²¹¹ Most ventilation rates for healthcare facilities are expressed as room air changes of filtered air per hour (ACH). Peak efficiency for particle removal in the air space occurs between 12 - 15 ACH.^{35, 240} Ventilation rates vary among the different patient-care areas of a healthcare facility (Appendix B).¹²⁰

Healthcare facilities generally use recirculated air.^{35, 120, 234, 241, 242} Fans create sufficient positive pressure to force air through the building duct work and adequate negative pressure to evacuate air from the conditioned space into the return duct work and/or exhaust, thereby completing the circuit in a sealed system (Figure 1). However, because gaseous

contaminants tend to accumulate as the air recirculates, a percentage of the recirculated air is exhausted to the outside and replaced by fresh outdoor air (usually a 60/40 mix of outdoor air/recirculated air).

In hospitals, filtered air is typically distributed from the ceiling, with return air collected from the ceiling on the other side of the room. In special situations in which the direction of air movement needs to be controlled (e.g., operating rooms, delivery rooms, catheterization laboratories, angiography rooms, HEPA-filtered rooms for immunosuppressed patients), the air is introduced from ceiling registers or diffusers near the center of the room, flows down to the patient-care zone, and is returned or exhausted through registers located at least 6 inches above the floor. Filtered air is introduced into negative-pressure, airborne infection isolation rooms (AII) above and near the doorway so that it passes through the breathing zone of workers and visitors before passing over the patient and being exhausted near the head of the bed, preferably from the side wall.^{4, 35}

Older hospitals with areas not served by central HVAC systems often use induction units (e.g., fan-coil units, heat-pump units) as the sole source of room ventilation. AIA guidelines for newly-installed systems stipulate that induction units shall be equipped with permanent (cleanable) or replaceable filters with a minimum efficiency of 68% weight arrestance.¹²⁰ These units may be used only as recirculating units; all outdoor air requirements must be met by a separate central air handling system with proper filtration, with a minimum of two outside air changes in general patient rooms.^{120, 243} If a patient room is equipped with an individual “through the wall” induction unit, the room should not be used as either AII or as PE.¹²⁰ These requirements, although directed to new installations, are also appropriate for existing settings. Induction units are prone to problems associated with excess condensation accumulating in drip pans and improper filter maintenance; healthcare facilities should clean or replace the filters in these units on a regular basis while the patient is out of the room.

Laminar airflow ventilation systems are designed to move air in a single pass, usually through a bank of HEPA filters either along a wall or in the ceiling, in a one-way direction through a clean zone with parallel streamlines. Laminar airflow can be directed vertically or horizontally; the unidirectional system optimizes airflow and minimizes air turbulence.^{63, 234} Delivery of air at a rate of 0.5 meters per second (90 ± 20 ft/min) helps to minimize opportunities for microorganism proliferation.^{63, 244, 245} Laminar airflow systems have been used in PE to help reduce the risk for healthcare-associated airborne infections such as aspergillosis in high-risk patients.^{63, 93, 246, 247} However, data that demonstrate a bona fide survival benefit for patients in PE with laminar airflow are lacking. Given the high cost of installation and apparent lack of benefit, the value of laminar airflow in this setting is questionable.^{9, 37} Few data support the use of laminar airflow systems elsewhere in a hospital.²⁴⁸

Positive and negative pressures refer to a pressure differential between two adjacent air spaces (e.g., rooms and hallways). Air flows away from areas or rooms with positive pressure, while air flows into areas with negative pressure. AII rooms are set at negative pressure to prevent airborne microorganisms in the room from entering hallways and corridors. PE rooms housing severely neutropenic patients are set at positive pressure to keep airborne pathogens in adjacent spaces or corridors from coming into and contaminating the airspace occupied by such high-risk patients. Self-closing doors are mandatory for both of these areas to help maintain the correct pressure differential.^{4, 6, 120} Older healthcare facilities may have variable pressure rooms (i.e., rooms in which the ventilation can be manually switched between positive- and negative pressure). These rooms are no longer permitted in the construction of new facilities,¹²⁰ and their use in existing facilities is discouraged because of difficulties in assuring the proper pressure differential, especially for the negative pressure setting, and the potential for error associated with switching the pressure differentials for the room. Engineering specifications for positive- or negative pressure rooms are given in Table 6.

Healthcare facilities must perform a risk assessment to determine the appropriate number of AII rooms (negative pressure) and/or PE rooms (positive pressure) to serve its patient population. The AIA guidelines require a certain number of AII rooms as a minimum.¹²⁰

Table 6. Engineered Specifications for Positive- and Negative Pressure Rooms^{35, 120}

	Positive Pressure Areas (e.g., PE)	Negative Pressure Areas (e.g., AII)
Pressure differentials	>2.5 Pa ^a (0.01" water gauge)	>2.5 Pa (0.01" water gauge)
Air changes per hour (ACH)	>12	≥12 (for renovation or new construction)
Filtration efficiency	Supply: 99.97% @ 0.3 μm DOP ^b Exhaust: None required	Supply: 90% (dust spot test) Exhaust: 99.97% @ 0.3 μm DOP ^b
Room airflow direction	Out to the adjacent area	In to the room
Clean-to-dirty airflow in room	Away from the patient (high-risk patient, immunosuppressed patient)	Towards the patient (airborne disease patient)
Ideal pressure differential	>8 Pa	>2.5 Pa

a. Pa = Pascal, a metric unit of measurement for pressure based on air velocity; 250 Pa equals 1.0 inch water gauge.

b. DOP = Dioctylphthalate particles of 0.3 μm diameter.

c. Used with permission of the publisher (reference 35).

In large healthcare facilities with central HVAC systems, sealed windows help to ensure the efficient operation of the system, especially with respect to creating and maintaining pressure differentials. Sealing the windows in PE areas will help to minimize the risk of airborne contamination from the outside. One outbreak of aspergillosis among immunosuppressed patients in a hospital was attributed in part to an open window in the unit during a time when both construction and a fire happened nearby; sealing the window prevented further entry of fungal spores into the unit from the outside air.¹¹¹ Additionally, all emergency exits (e.g., fire escapes, emergency doors) in PE wards should be kept closed (except during emergencies) and equipped with alarms.

e. Infection Control Impact of HVAC System Maintenance and Repair

A failure or malfunction of any component of the HVAC system may subject patients and staff to discomfort and exposure to airborne contaminants. Little information is available from formal studies on the infection control implications of a complete air-handling system failure or shutdown for maintenance. Most experience has been derived from infectious disease outbreaks and adverse outcomes among high-risk patients when HVAC systems are poorly maintained. Table 7 summarizes potential ventilation hazards, consequences, and correction measures.

AIA guidelines prohibit United States hospitals and surgical centers from shutting down their HVAC systems for purposes other than required maintenance, filter changes, and construction.¹²⁰ Airflow can be reduced, but sufficient supply, return, and exhaust must be provided to maintain required pressure relationships when the space is not occupied. This can be accomplished with special drives on the air-handling units (i.e., a Variable Air Ventilation [VAV] system).

Microorganisms proliferate in environments wherever air, dust, and water are present, and air-handling systems can be ideal environments for microbial growth.³⁵ Properly engineered HVAC systems require routine maintenance and monitoring in order to provide acceptable indoor air quality efficiently and to minimize conditions that favor the proliferation of healthcare-associated pathogens.^{35, 241} Performance monitoring of the system includes determining pressure differentials across filters, regular inspection of system filters, DOP testing of HEPA filters, testing of low- or medium efficiency filters, and manometer tests for positive- and negative-pressure areas in accordance with nationally recognized standards, guidelines, and manufacturers' recommendations. The use of hand-held calibrated equipment that can provide a numerical reading on a daily basis is preferred for engineering purposes.^{249, 250} Several methods that provide a visual, qualitative measure of pressure differentials include smoke-tube tests, or placing flutter strips, ping-pong balls, or tissue in the air stream.

Preventive filter and duct maintenance (e.g., cleaning ductwork vents, replacing filters as needed, properly disposing spent filters into plastic bags immediately upon removal) is important to prevent potential exposures of patients and staff during HVAC system shut-down. Additionally, a malfunction of the air-intake system can overburden the filtering system and permit aerosolization of fungal pathogens. Keeping the intakes free from bird droppings, especially those

from pigeons, helps to minimize the concentration of fungal spores entering from the outside.⁹⁸

Accumulation of dust and moisture within HVAC systems increases the risk of spread of healthcare-associated environmental fungi and bacteria. Clusters of infections due to *Aspergillus* spp., *Pseudomonas aeruginosa*, *Staphylococcus aureus*, and *Acinetobacter* spp. have been linked to poorly maintained and/or malfunctioning air conditioning systems.^{68, 159, 251, 252} Efforts to limit excess humidity and moisture in the infrastructure and on air stream surfaces in the HVAC system can minimize the proliferation and dispersion of fungal spores and waterborne bacteria throughout the indoor air.^{253 - 255} Within the HVAC system, water is present in water-wash units, humidifying boxes, or cooling units. The dual-duct system may also create conditions of high humidity and excess moisture that favor fungal growth in drain pans as well as in fibrous insulation material which becomes damp as a result of the humid air passing over the hot stream and condensing. All intake air should be dehumidified to avoid condensation when the air is mixed.

If moisture is present in the HVAC system, it is important to avoid periods of stagnation, such as would occur if the system is temporarily shut down. Bursts of organisms tend to be released upon system start-up which may increase the risk of airborne infection.²⁰² Proper engineering of the HVAC system is critical to preventing dispersal of airborne organisms. Endophthalmitis due to *Acremonium kiliense* infection following cataract extraction in an ambulatory surgical center was traced to aerosols derived from the humidifier water in the ventilation system.²⁰² The organism proliferated because the ventilation system was turned off routinely when the center was not in operation and the air was filtered before humidification, but not afterwards.

Most healthcare facilities have contingency plans in case of disruption of HVAC services, including back-up power generators that maintain the ventilation system in high-risk areas (e.g., operating rooms, intensive care units, negative- or positive-pressure rooms, transplantation and oncology units). Alternative generators are required to engage within 10 seconds of a loss of main power. If the ventilation system is out of service such that the indoor air becomes stagnant, sufficient time must be allowed to clean the air and re-establish the appropriate number of ACH once the HVAC system comes back on line. Air filters may also need to be changed because reactivation of the system can dislodge large amounts of dust and create a transient burst of fungal spores.

Duct cleaning in healthcare facilities has benefits in terms of system performance, but its usefulness for infection control has not been conclusively determined. Duct cleaning typically involves using specialized tools to dislodge dirt and a high-powered vacuum cleaner to clean out debris. Some duct-cleaning services also apply chemical biocides or sealants to the inside surfaces of ducts to minimize fungal growth and prevent the release of particulate matter. Although infrequent cleaning of the exhaust ducts in AII areas has been documented to be a cause of diminishing negative pressure and a decrease in the air exchange rates,²¹⁰ there are no data to indicate that duct cleaning, beyond what is recommended for optimal performance, improves indoor air quality or reduces the risk of infection. Exhaust return systems should be cleaned as part of routine system maintenance. Duct cleaning has not been shown to prevent any health problems,²⁵⁶ and U.S. Environmental Protection Agency (EPA) studies indicate that airborne particulate levels do not increase as a result of dirty air ducts, nor do they diminish after cleaning, presumably because much of the dirt inside air ducts adheres to duct surfaces and does not enter the conditioned space.²⁵⁶ Additional research is needed to determine if air duct contamination can significantly increase the airborne infection risk in general areas of healthcare facilities.

Table 7. Ventilation Hazards in Healthcare Facilities That May Be Associated with Increased Potential of Airborne Disease Transmission³⁵

Problem	Consequences	Possible Solutions
Water-damaged building materials (18)	Water leaks can soak wood, wall board, insulation, wall coverings, ceiling tiles, and carpeting. All of these materials can provide microbial habitat when wet. This is especially true for fungi growing on gypsum board.	<ol style="list-style-type: none"> 1. Replace water-damaged materials. 2. Incorporate fungistatic compounds into building materials in areas at risk for moisture problems. 3. Test for all moisture and dry in less than 72 hours. Replace if the material cannot dry within 72 hours.
Filter bypasses (17)	Rigorous air filtration requires air flow resistance. Air stream will elude filtration if openings are present because of filter damage or poor fit.	<ol style="list-style-type: none"> 1. Use pressure gauges to ensure that filter is performing at proper static pressure. 2. Make ease of installation and maintenance criteria for filter selection. 3. Properly train maintenance personnel in HVAC issues. 4. Design system with filters down-stream from fans. 5. Avoid water on filters or insulation.
Improper fan setting (257)	Air must be delivered at design volume to maintain pressure balances. Airflow in special vent rooms reverses.	<ol style="list-style-type: none"> 1. Routinely monitor air flow and pressure balances throughout critical parts of HVAC system. 2. Minimize or avoid using rooms that switch between positive- and negative pressure.
Ductwork disconnections (258)	Dislodged or leaky supply duct runs can spill into or leaky returns may draw from hidden areas. Pressure balance will be interrupted, and infectious material may be disturbed and entrained into hospital air supply.	<ol style="list-style-type: none"> 1. Design a ductwork system that is easy to access, maintain, and repair. 2. Train maintenance personnel to regularly monitor air flow volumes and pressure balances throughout the system. 3. Test critical areas for appropriate airflow.
Air flow impedance (209)	Debris, structural failure, or improperly adjusted dampers can block duct work and prevent designed air flow.	<ol style="list-style-type: none"> 1. Design and budget for a duct system that is easy to inspect, maintain, and repair. 2. Alert contractors to use caution when working around HVAC system during the construction phase. 3. Regularly clean exhaust grills. 4. Provide monitoring for special ventilation areas.
Open windows (96, 240)	Open windows can alter fan induced pressure balance and allow dirty-to-clean air flow.	<ol style="list-style-type: none"> 1. Use sealed windows. 2. Design HVAC system to deliver sufficient outdoor dilution ventilation. 3. Ensure that OSHA indoor air quality standards are met.
Dirty window air conditioners (96, 259)	Dirt, moisture, and bird droppings can contaminate window air conditioners, which can then introduce infectious material into hospital room.	<ol style="list-style-type: none"> 1. Eliminate such devices in plans for new construction. 2. Where they must be used, make sure that they are routinely cleaned and inspected.
Inadequate filtration (260)	Infectious particles may pass through filter into vulnerable patient areas.	<ol style="list-style-type: none"> 1. Specify appropriate filters during new construction design phase. 2. Make sure that HVAC fans are sized to overcome pressure demands of filter system. 3. Inspect and test filters for proper installation.
Maintenance disruptions (261)	Fan shut-offs, dislodged filter cake material contaminates downstream air supply, and drain pans. Compromises airflow in special ventilation areas.	<ol style="list-style-type: none"> 1. Be sure to budget for rigorous maintenance schedule when designing a facility. 2. Design system for easy maintenance. 3. Ensure good communication between engineering and maintenance personnel. 4. Institute an ongoing training program for all involved staff members.

Table 7 (continued) Ventilation Hazards

Problem	Consequences	Possible Solutions
Excessive moisture in the HVAC system (120)	Chronically damp internal lining of the HVAC system, excessive condensate, drip pans with stagnant water	<ol style="list-style-type: none"> 1. Duct humidifiers should be located upstream of the final filters 2. There should be a means to remove water from the system. 3. Monitor humidity; all duct takeoffs should be downstream of the humidifiers so that moisture is absorbed completely. 4. Use steam humidifiers in the HVAC system.
Duct contamination (18, 262)	Debris is released during maintenance or cleaning.	<ol style="list-style-type: none"> 1. Provide point-of-use filtration in the critical areas. 2. Design air handling system with insulation on the exterior of the ducts. 3. No fibrous sound attenuators. 4. Decontaminate or encapsulate contamination.

a. Used with permission of the publisher (reference 35).

4. Construction, Renovation, Remediation, Repair, and Demolition

a. General Information

Environmental disturbances caused by construction and/or renovation and repair activities (e.g., removing ceiling tiles, running cables through the ceiling, structural repairs) in and around healthcare facilities markedly increase the airborne *Aspergillus* spp. spore counts in the indoor air of such facilities, thereby increasing the risk for healthcare-associated aspergillosis among high-risk patients. Although one case of healthcare-associated aspergillosis is often difficult to link to a specific environmental exposure, the occurrence of temporarily clustered cases increase the likelihood that an environmental source within the facility may be identified and corrected.

Construction, renovation, repair, and demolition activities in healthcare facilities require substantial planning and coordination to minimize the risk of airborne infection both during projects and after their completion. Several organizations and experts have endorsed a multi-disciplinary team approach to coordinate the various stages of construction activities (e.g., project inception, project implementation, final walk-through, and completion).^{120, 241, 242, 263 - 266} Table 8 lists suggested members of a multi-disciplinary team, team functions, and responsibilities.

Table 8. Suggested Members and Functions of a Multi-Disciplinary Coordination Team for Construction, Renovation, Repair, and Demolition Projects

<i>Members</i>
Infection control personnel, including hospital epidemiologists
Laboratory personnel
Facility administrators / representatives
Director of engineering
Risk management personnel
Directors of specialized programs (e.g., transplantation, oncology, intensive care unit programs)
Employee safety / regulatory affairs personnel
Environmental services
Information systems personnel
Construction administrators / representatives
Architects
Project managers
Contractors
Industrial hygienists

<i>Functions and Responsibilities</i>
Coordinate members' input in developing a comprehensive project management plan.
Conduct a risk assessment of the project to determine potential hazards to susceptible patients.
Prevent unnecessary exposures of patients, visitors, and staff to infectious agents.
Oversee all infection control aspects of construction activities.
Establish site-specific infection control protocols for specialized areas.
Provide education about the infection control impact of construction to staff and construction workers.
Ensure compliance with technical standards, contract provisions, and regulations.
Establish a mechanism to address and correct problems quickly.
Develop contingency plans for power failures, water supply disruptions, fires, and emergency response.
Provide a water damage management plan (including drying protocols) for handling water intrusion from floods, leaks, and condensation.
Develop a plan for structural maintenance.

Education of maintenance and construction workers, healthcare staff charged with the care of with high-risk patients, and persons responsible for controlling indoor air quality can help to minimize dust and moisture intrusion from construction sites into high-risk patient care areas.^{120, 242, 263, 265 - 268} Visual and printed educational materials should be provided as appropriate in the language of the workers. Staff and construction workers also need to be aware of the potentially catastrophic consequences of dust and moisture intrusion when an HVAC system or water system fails during construction or repair; action plans to deal quickly with these emergencies should be developed in advance and kept on file. Incorporation of specific standards into construction contracts may help to prevent departures from recommended practices as projects progress. Establishing specific lines of communication is important to address problems (e.g., dust control, indoor air quality, noise levels, vibrations), resolve complaints, and keep projects moving toward completion. Healthcare facility staff should develop a mechanism to monitor worker adherence to infection control guidelines on a daily basis in and around the construction site for the duration of the project.

b. Preliminary Considerations

Three major topics to consider before initiating any construction or repair activity are: 1) design and function of the new structure or area; 2) assessment of environmental risks for airborne disease and opportunities for prevention; and 3) measures to contain dust and moisture during construction or repairs. Table 9 provides a checklist of design and function considerations to ensure that a planned structure or area can be easily serviced and maintained for environmental infection control.^{17, 241, 263, 265 - 267} Specifications for the construction, renovation, remodeling, and

maintenance of healthcare facilities are outlined in the AIA document, *Guidelines for Design and Construction of Hospitals and Health Care Facilities*.^{120, 265}

Table 9. Construction Design and Function Considerations for Environmental Infection Control

Location of sinks and handwashing product dispensers
Types of faucets (aerated vs. non-aerated)
Air handling systems engineered for optimal performance and easy maintenance and repair
ACH and pressure differentials to accommodate special patient care areas
Location of fixed sharps containers
Types of surface finishes (non-porous vs. porous)
Well-caulked walls with minimal seams
Location of adequate storage and supply areas
Appropriate location of medicine preparation areas (e.g., ≥ 3 ft. from a sink)
Appropriate location and type of ice machines (preferably single-use- vs. chest-type dispensers)
Appropriate materials for sinks and wall coverings
Appropriate traffic flow (no “dirty” movement through “clean” areas)
Isolation rooms with anterooms as required
Appropriate flooring (e.g., seamless floors in dialysis units)
Sensible use of carpeting (e.g., no carpeting in special care areas or areas likely to become wet)^a
Convenient location of soiled utility areas
Properly engineered areas for linen services and solid waste management
Location of main generator to minimize the risk of system failure from flooding or other emergency)
Installation guidelines for sheetrock

a. Although bacteria and fungi can be recovered in great numbers from carpet, its use has not been shown to be a consistent risk for healthcare-associated infections in immunocompromised patient-care areas. Use of carpet cleaning methods (e.g., “bonneting”) that disperse microorganisms into the air of these special patient care areas, however, may increase the risk of airborne infection.¹¹¹

Proactive strategies can help prevent environmentally-mediated airborne infections in healthcare facilities during demolition, construction, and renovation. The potential presence of dust and moisture and their contribution to healthcare-associated infections must be critically evaluated early in the planning of any demolition, construction, renovation, and repairs.^{120, 241, 242, 263, 264, 266 - 269} Consideration must extend beyond dust generated by major projects to include dust that can become airborne if disturbed during routine maintenance and minor renovation activities (e.g., exposure of ceiling spaces for inspection; installation of conduits, cable, or sprinkler systems; rewiring; structural repairs or replacement).^{263, 266, 267} Other projects that can compromise indoor air quality include construction and repair jobs that inadvertently allow substantial amounts of raw, unfiltered outdoor air to enter the facility (e.g., repair of elevators and elevator shafts) and activities that dampen any structure, area, or item made of porous materials or characterized by cracks and crevices (e.g., sink cabinets in need of repair, carpets, ceilings, floors, walls, vinyl wall coverings, upholstery, drapes, and countertops).^{18, 263, 267} Molds grow and proliferate on these surfaces should these materials become and remain wet.^{21, 120, 242, 260, 263, 270} Scrubbable materials are preferred for use in patient-care areas.

Containment measures for dust and/or moisture control are dictated by the location of the construction site. Outdoor demolition and construction require actions to keep dust and moisture out of the facility (e.g., sealing windows and vents, keeping doors closed or sealed). Containment of dust and moisture generated from construction inside a facility requires barrier structures (either pre-fabricated or constructed of more durable materials as needed) and engineering controls to clean the air in and around the construction/repair site.

c. Infection Control Risk Assessment

A risk assessment conducted before initiating demolition, construction, or renovation activities can identify potential exposures of susceptible patients to dust and moisture and determine the need for dust and moisture containment measures. This assessment centers on the type and extent of the construction or repairs in the work area but may also need to consider adjacent patient-care areas, supply storage, and areas on levels above and below the proposed project.

Knowledge of the airflow patterns and pressure differentials will help to minimize or eliminate the inadvertent dispersion of dust that could contaminate air space, patient-care items, and surfaces.^{57, 271, 272} During long-term projects, providing temporary essential services (e.g., toilet facilities, vending machines) to construction workers within the site will help to minimize traffic in and out of the area. The type of barrier systems necessary for the scope of the project must be defined.^{12, 120, 242, 269, 273}

Depending on the location and extent of the construction, patients may need to be relocated to other areas in the facility not affected by construction dust.^{51, 274} This is especially important when construction takes place within units housing immunocompromised patients, severely neutropenic patients, or patients on corticosteroid therapy. Advance assessment of high-risk locations and planning for the possible transport of patients to other departments can minimize delays and waiting time in hallways.⁵¹ Hospitals may provide immunocompromised patients with respiratory protection devices for use outside their rooms, although this has not been evaluated for preventing exposure to fungal spores. Protective respirators (i.e., N95) appeared to be well tolerated by patients in one recent study.²⁷²

Surveillance activities should augment preventive strategies during construction projects.^{3, 4, 20, 110, 275, 276} By determining baseline levels of healthcare-acquired airborne and waterborne infections, infection control staff can monitor changes in infection rates and patterns during and immediately after construction, renovations, or repairs.³

d. Air Sampling

(See also **F. Environmental Sampling** later on in Part I for additional basic information.)

Air sampling in healthcare facilities may be used both during periods of construction and on a periodic basis to determine indoor air quality, efficacy of dust control measures, or air-handling system performance via parametric monitoring. Parametric monitoring consists of measuring the physical performance of the HVAC system in accordance with the system manufacturer's specifications. A periodic assessment of the system can give assurance of proper ventilation, especially for special-care areas and operating rooms (e.g., airflow direction and pressure, ACH, filter efficiency).²⁷⁷

Air sampling is used to detect aerosols (particles or microorganisms). Particulate sampling (i.e., total numbers and size range of particulates) is a practical method for evaluating the infection-control performance of the HVAC system, with an emphasis on filter efficiency in removing respirable particles (<5µm diameter) or larger particles from the air. Particle size is reported in terms of the mass median aerodynamic diameter (MMAD), while count median aerodynamic diameter (CMAD) is useful with respect to particle concentrations.

Particle counts in a given air space within the healthcare facility should be evaluated against counts obtained in a comparison area. Particle counts indoors are commonly compared with the particulate levels of the outdoor air. This approach determines the "rank order" air quality from "dirty" (i.e., the outdoor air) to "clean" (i.e., air filtered through high-efficiency filters [90% - 95% filtration]) to "cleanest" (i.e., HEPA-filtered air).²⁷⁷ Comparisons from one indoor area to another may also provide useful information about the magnitude of an indoor air quality problem. Presently, rank-order comparisons among clean, highly-filtered areas and dirty areas and/or outdoors has been suggested as one way to interpret sampling results in the absence of air quality and action level standards.^{35, 278}

In addition to verifying filter performance, particle counts can help determine if barriers and efforts to control dust dispersion from construction are effective. This type of monitoring is helpful when performed at various times and barrier perimeter locations during the project. Gaps or breaks in the barriers' joints or seals can then be identified and repaired. With respect to occupational health, the American Conference of Governmental Industrial Hygienists (ACGIH) has set a threshold limit value-time weighted average (TLV®-TWA) of 10 mg/m³ for nuisance dust that contains no asbestos and <1% crystalline silica.²⁷⁹ Alternatively, OSHA has set permissible exposure limits (PELs) for inert or nuisance dust as follows: respirable fraction at 5 mg/m³ and total dust at 15 mg/m³.²⁸⁰ Although these standards are not measures of a bioaerosol, they are used for indoor air quality assessment in occupational settings and may be useful criteria in construction areas. Application of ACGIH guidance to healthcare settings has not been standardized, but particulate counts in healthcare facilities are likely to be well below this threshold value and approaching clean-room standards in care areas such as operating rooms.¹⁰⁰

Particle counters and anemometers are used in particulate evaluation. The anemometer measures air flow velocity, which can be used to determine sample volumes. Particulate sampling usually does not require microbiology laboratory services for the reporting of results.

Microbiologic sampling of air in healthcare facilities remains controversial because of currently unresolved technical limitations and the need for substantial laboratory support (Table 10).

The most significant technical limitation of air sampling for airborne fungal agents is the lack of standards linking fungal spore levels with infection rates. Despite this limitation, several healthcare institutions have opted to use microbiologic sampling when construction projects are anticipated and/or underway in efforts to assess the safety of the environment for the immunocompromised patients.^{35, 278} From a practical standpoint, microbiologic air sampling should be limited to assays for airborne fungi; of those, the thermotolerant fungi (i.e., those capable of growing at 35°C - 37°C [95°F - 98.6°F]) are of particular concern because of their pathogenicity in immunocompromised hosts.³⁵ Use of selective media (e.g., Sabourauds, inhibitory mold agar) helps with the initial identification of recovered organisms.

Microbiologic sampling for fungal spores performed as part of various airborne disease outbreak investigations has also been problematic.^{18, 49, 106, 111, 112, 278} The precise source of a fungus is often difficult to trace with certainty, and sampling conducted after exposure may neither reflect the circumstances that were linked to infection nor distinguish between healthcare-acquired and community-acquired infections. Because fungal strains may fluctuate rapidly in the environment, healthcare-acquired *Aspergillus* spp. infection cannot be confirmed or excluded if the infecting strain is not found in the healthcare setting.²⁷⁶ The use of sensitive molecular typing methods (e.g., randomly amplified polymorphic DNA (RAPD) techniques or more recently a DNA fingerprinting technique that detects restriction fragment length polymorphisms in fungal genomic DNA) to identify strain differences among *Aspergillus* spp., however, is increasing in importance in epidemiologic investigations of healthcare-acquired fungal infection.^{68, 110, 250, 275, 276, 281 - 285} During case cluster evaluation, microbiologic sampling may provide an isolate from the environment for molecular typing and comparison with patient isolates. It may be prudent for the clinical laboratory to save *Aspergillus* spp. isolated from invasive disease cases for these purposes.

Sedimentation methods using settle plates and volumetric sampling methods using solid impactors are commonly employed when sampling air for bacteria and fungi. Settle plates have been used by numerous investigators to detect airborne bacteria or to measure air quality during medical

Table 10. Unresolved Issues Associated with Microbiologic Air Sampling^{35, 100, 215, 278, 286}

Lack of standards linking fungal spore levels with infection rates (i.e., no safe level of exposure)
Lack of standard protocols for testing (e.g., sampling intervals, number / location of samples)
Need for substantial laboratory support
New, complex PCR analytical methods
Unknown incubation period for *Aspergillus* spp. infection
Variability of sampler readings
Sensitivity of the sampler used (i.e., the volumes of air sampled)
Lack of details in the literature about describing sampling circumstances (e.g., unoccupied rooms vs. ongoing activities, expected fungal concentrations, rate of outdoor air penetration)
Lack of correlation between fungal species and strains from the environment and clinical specimens
Confounding variables with high-risk patients (e.g., visitors, time spent outside of PE without protective respiratory equipment)
Need for determination of ideal temperature for incubating fungal cultures (35°C [95°F] is preferred)

procedures (e.g., during surgery).^{17, 60, 97, 149, 159, 276} Settle plates, because they rely on gravity during sampling, tend to select for larger particles and lack sensitivity for respirable particles (e.g., individual fungal spores), especially in highly-filtered environments, and thus are considered impractical for general use.^{35, 278, 287 - 290} Settle plates, however, may

detect fungi aerosolized during medical procedures (e.g., during wound dressing changes), as described in a recent outbreak of aspergillosis among liver transplant patients.²⁹¹

The use of slit or sieve impactor samplers capable of collecting large volumes of air in short periods of time are needed to detect low numbers of fungal spores in highly-filtered areas.^{35, 278} In some outbreaks, aspergillosis cases have occurred when fungal spore concentrations in PE ambient air ranged as low as 0.9 - 2.2 colony-forming units per cubic meter (CFU/m³) of air.^{18, 94} Based on the expected spore counts in the ambient air and the performance parameters of various types of volumetric air samplers, investigators of a recent aspergillosis outbreak have suggested that an air volume of at least 1000 L (1 m³) should be considered when sampling highly filtered areas.²⁷² Investigators have also suggested limits of 15 CFU/m³ for gross colony counts of fungal organisms and <0.1 CFU/m³ for *Aspergillus fumigatus* and other potentially opportunistic fungi in heavily filtered areas (≥ 12 ACH and filtration of $\geq 99.97\%$ efficiency).¹²⁰ There has been no reported correlation of these values with the incidence of healthcare-associated fungal infection rates.

Air sampling in healthcare facilities, whether used to monitor air quality during construction, to verify filter efficiency, or to commission new space prior to occupancy, depends on careful notation of the circumstances of sampling. Most air sampling is performed under undisturbed conditions. However, when the air is sampled during or after human activity (e.g., walking, vacuuming), a higher number of airborne microorganisms is detected.²⁸⁶ The contribution of human activity to the significance of air sampling and the impact on healthcare-associated infection rates remain to be defined. Comparing microbiologic sampling results from a target area (e.g., an area of construction) to those from an unaffected location in the facility can provide information about distribution and concentration of potential airborne pathogens. A comparison of microbial species densities of outdoor air to those obtained from indoor air has been used to help pinpoint fungal spore bursts. Fungal spore densities in outdoor air are variable, although the degree of variation with the seasons appears to be more dramatic in the United States than in Europe.^{92, 276, 292}

Particulate and microbiologic air sampling have been used when commissioning new HVAC system installations, but this is particularly important for newly constructed or renovated PE or operating rooms. Particulate sampling is used as part of a battery of tests to determine if a new HVAC system is performing to specifications for filtration and the proper number of ACH.^{258, 277, 293} Microbiologic air sampling, however, remains controversial in this application, as there are no standards for comparison purposes. If performed, it should be limited to determining the density of fungal spores per unit volume of air space. High numbers of spores may indicate contamination of air handling system components prior to installation, or a system deficiency when culture results are compared to known filter efficiencies and rates of air exchange.

e. External Demolition and Construction

External demolition and dirt excavation generate considerable dust and debris that can contain airborne microorganisms. In one study, peak concentrations in outdoor air at grade level and HVAC intakes during site excavation averaged 20,000 CFU/m³ for all fungi and 500 CFU/m³ for *Aspergillus fumigatus*, compared with 19 CFU/m³ and 4 CFU/m³, respectively, in the absence of construction.²⁷⁰ Important issues to review prior to demolition include:^{120, 241, 242, 263, 266, 267, 270, 294} 1) proximity of the air intake system to the work site; 2) adequacy of window seals and door seals; 3) proximity of areas frequented by immunocompromised patients; and 4) location of the underground water pipes. Strategies for minimizing the intrusion of dust and moisture are summarized in Table 11.

Preventing the entry of outside dust into the HVAC system is crucial. Facility engineers should be consulted about the potential impact of shutting down the system or increasing the filtration.

Table 11. Strategies to Reduce Dust and Moisture Contamination During External Demolition and Construction

Item	Recommendation
Demolition site	U Shroud the site if possible to reduce environmental contamination.
Adjacent air intakes	U Seal off affected intakes if possible, or move if funds permit.
HVAC system	U Consult with facility engineer about pressure differentials, air recirculation options; keep facility air pressure positive relative to outside air.
Filters	U Ensure that filters are properly installed; change roughing filters frequently to prevent dust build-up on high-efficiency filters.
Windows	U Sealed and caulked to prevent entry of airborne fungal spores.
Doors	U Keep closed as much as possible; do not prop open; seal and caulked unused doors (i.e., those that are not designated as emergency exits); use tacky mats at outside entrances.
Water pipes	U Note location relative to construction area to prevent intrusion of dust into water systems. ^a
Rooftops	U Avoid during active demolition/construction.
Dust generation	U Mist the area with water to minimize dust.
Immunocompromised patients	U Use respiratory barriers (e.g., N95) to prevent airborne infections from demolition dust; use walk-ways protected from demolition/construction sites; avoid outside areas close to these sites; avoid rooftops.
Truck traffic	U Reroute if possible, or arrange for frequent street cleaning.
Education/awareness	U Encourage reporting of incidents associated with construction.

a. Contamination of water pipes during demolition activities has been associated with healthcare-associated transmission of *Legionella*.²⁹⁴

Selected air handlers, especially those located close to excavation sites, may have to be shut off temporarily to keep from overloading the system with dust and debris. Care is needed to avoid significant facility-wide reductions in pressure differentials that may cause the building to become negatively pressured relative to the outside. To prevent excessive particulate overload and subsequent reductions in effectiveness of intake air systems that cannot be shut off temporarily, air filters must be inspected frequently for proper installation and function. Excessive dust penetration can be avoided if recirculated air is maximally utilized while outdoor air intakes are shut down. Scheduling demolition and excavation during the winter, when *Aspergillus* spp. spores may be present in lower numbers, can help, although seasonal variations in spore density differ around the world.^{92, 276, 292} Dust control can be managed by misting the dirt and debris during heavy dust-generating activities. To decrease the amount of aerosols from excavation and demolition projects, nearby windows, especially in areas with immunocompromised patients, can be sealed and window and door frames caulked or weather-stripped to prevent dust intrusion.^{50, 290, 295} Monitoring for adherence to these control measures throughout demolition or excavation is crucial. Diverting pedestrian traffic away from the construction sites decreases the amount of dust tracked back into the healthcare facility and minimizes exposure of high-risk patients to environmental pathogens.

f. Internal Demolition, Construction, Renovations, and Repairs

The focus for infection control during interior construction and repairs is containment of dust and moisture. This objective is achieved by: 1) educating construction workers about the importance of control measures; 2) preparing the site; 3) notifying and issuing advisories for staff, patients, and visitors; 4) moving staff and patients and relocating patients as needed; 5) issuing standards of practice and precautions during activities and maintenance; 5) monitoring for adherence to control measures during construction and providing prompt feedback about lapses in control; 6) monitoring HVAC performance; 7) implementing daily clean-up, terminal cleaning and removal of debris upon completion; and 8) maintaining the water system after construction.

Physical barriers to contain smoke and dust will confine disbursed fungal spores to the construction zone.^{269, 273, 296, 297} The type of barrier required depends on the project's scope and duration and on local fire codes. Short-term projects

that result in minimal dust dispersion (e.g., installation of new cables or wiring above ceiling tiles) require only portable plastic enclosures with negative pressure and HEPA filtration of the exhaust air from the enclosed work area. The placement of a portable industrial-grade HEPA filter device (300 - 800 ft³/min.) adjacent to the work area will help to remove fungal spores, but its efficacy is dependent on the supplied ACH and size of the area. If the project is more extensive than a repair job but still is considered a short-term undertaking, then dust-abatement, fire-resistant plastic curtains (e.g., Visqueen®) may be adequate. These should be completely airtight and sealed from ceiling to floor with overlapping curtains;^{266, 267, 298} holes, tears, or other perforations should be repaired promptly with tape. A portable industrial-grade HEPA filter unit on continuous operation may be needed within the contained area, with the filtered air exhausted to the outside of the work zone. Patients should not remain in the room when dust-generating activities are performed.

More elaborate barriers are indicated for projects of long duration that generate moderate to large amounts of dust. These barrier structures typically consist of rigid, noncombustible walls constructed from sheet rock, drywall, plywood, or plaster board and covered with sheet plastic (e.g., Visqueen®). Barrier requirements to prevent the intrusion of dust into patient-care areas include: 1) installing a plastic dust abatement curtain before construction of the rigid barrier; 2) sealing and taping all joint edges including the top and bottom; 3) extending the barrier from floor to floor, which takes into account the space (approximately 2 - 8 ft.) above the finished, lay-down ceiling; and 4) fitting or sealing any temporary doors connecting the construction zone to the adjacent area. Table 12 lists some of the various construction and repair activities that require the use of temporary or durable barriers.

Table 12. Construction/Repair Projects That Require Barrier Structures^{120, 242, 263, 266, 267}

Demolition of walls, wallboard, plaster, ceramic tiles, ceiling tiles, ceilings
 Removal of flooring and carpeting, windows and doors, casework
 Working with sinks and plumbing that could result in aerosolization of water in high-risk areas
 Exposure of ceiling spaces for installation of conduits, rewiring
 Crawling into ceiling spaces for inspection (but not for visual inspection only)
 Demolition, repair, or construction of elevator shafts
 Repairing water damage

Dust and moisture abatement and control rely primarily on the impermeable barrier containment approach; as construction continues, numerous opportunities can lead to dispersion of dust to other areas of the healthcare facility. This is especially true for PE for neutropenic patients. Infection control measures which augment the use of barrier containment are outlined in Table 13. This table presents a generalized approach of managing internal construction and repair projects through the entire process.

Table 13. Infection Control Measures for Internal Construction and Repair Projects^{19, 51, 67, 80, 106, 120, 241, 242, 263, 266 - 268, 270, 274, 298 - 301}

Infection Control Measure	Steps Toward Implementation
Get ready for the project	<ol style="list-style-type: none"> 1. Use a multi-disciplinary team approach to incorporate infection control into the project. 2. Conduct the risk assessment, preliminary walk-through with project managers and staff.
Educate staff and construction workers	<ol style="list-style-type: none"> 1. Educate staff and construction workers about the importance of adhering to infection control measures during the project. 2. Provide educational materials in the language of the workers.
Issue hazard and warning notices	<ol style="list-style-type: none"> 1. Post signs to identify construction areas, potential hazards. 2. Mark detours to avoid the work area.
Relocate high-risk patients as needed, especially if the construction is in or adjacent to a PE area.	<ol style="list-style-type: none"> 1. Identify target patient populations for relocation based on the risk assessment. 2. Arrange for the transfer in advance to avoid delays. 3. At-risk patients should wear protective respiratory equipment (e.g., N95 respirator).
Establish alternative traffic patterns for staff, patients, visitors, and construction workers	<ol style="list-style-type: none"> 1. Determine appropriate alternate routes from the risk assessment. 2. Designate areas (e.g., hallways, elevators, entrances/exits) for construction worker use. 3. Do not transport patients on the same elevator with construction materials and debris.
Erect appropriate barrier containment	<ol style="list-style-type: none"> 1. Use prefabricated plastic units or plastic sheeting for short-term projects which would generate minimal dust. 2. Use durable rigid barriers for ongoing, long-term projects.
Establish proper ventilation	<ol style="list-style-type: none"> 1. Shut off return air vents in construction zone if possible and seal around grilles. 2. Exhaust air and dust to the outside if possible. 3. If recirculated air from the construction zone unavoidable, use a pre-filter and a HEPA filter before the air returns to the HVAC system. 4. Set pressure differentials so that the contained work area is under negative pressure. 5. Use airflow monitoring devices to verify the direction of the air pattern. 6. Exhaust air and dust to the outside if possible. 7. Monitor temperature, ACH, and humidity levels (humidity < 65%). 8. Use portable, industrial grade HEPA filters in the adjacent area and/or the construction zone for additional ACH. 9. Keep windows closed if possible.
Control debris	<ol style="list-style-type: none"> 1. When replacing filters, place the old filter in a bag prior to transport and disposal. 2. Clean the construction zone daily or more often as needed. 3. Designate removal route for small quantities of debris. 4. Mist debris and cover disposal carts before transport. 5. Designate an elevator for construction crew use. 6. Use window chutes and negative pressure equipment for removal of larger pieces of debris while maintaining pressure differentials. 7. Time debris removal to periods when patient exposure to dust is minimal.
Control dust	<ol style="list-style-type: none"> 1. Monitor the construction area daily for compliance with the infection control plan. 2. Protective outer clothing for construction workers should be removed before entering clean areas. 3. Use tacky mats within the construction zone at the entry; cover sufficient area so that both feet make contact with the mat while walking through the entry. 4. Construct an anteroom as needed where coveralls can be removed. 5. Use wet-mop cleaning of the construction zone and all areas used by construction workers. 6. Provide temporary essential services (e.g., toilets, vending machines) in the construction zone. 7. Damp-wipe tools if removed from the construction zone or left in the area. 8. Ensure that the barriers remain well sealed; use particle sampling as needed. 9. Ensure that the clinical laboratory is free from dust contamination.

(Continued next page)

Table 13 (continued). Infection Control Measures for Internal Construction

Infection Control Measure	Steps Toward Implementation
Control water damage	<ol style="list-style-type: none"> 1. Make provisions for dry storage of building materials. 2. Do not install wet, porous building materials (i.e., sheet rock). 3. Replace water damaged porous building materials if they cannot be completely dried out within 72 hours.
Complete the project	<ol style="list-style-type: none"> 1. Flush the mains water system to clear dust contaminated lines. 2. Terminally clean the construction zone before the barriers are removed. 3. Check for visible mold and mildew and remove if present. 4. Do not accept ventilation deficiencies in special care areas. 5. Verify appropriate ventilation parameters for the new area as needed. 6. Clean or replace HVAC filters using proper dust containment procedures. 7. Remove the barriers. 8. Ensure designated air balance in the OR and protective environments before occupancy. 9. Commission the space as indicated, especially for the OR and protective environments.

Dust control measures for the clinical laboratories are an important part of the infection control strategy during hospital construction or renovation. Pseudofungemia clusters attributed to *Aspergillus* spp. and *Penicillium* spp. have been linked to improper airflow patterns and construction adjacent to the laboratory, intrusion of dust and spores into a biological safety cabinet from construction activity immediately next to the cabinet resulted in a cluster of cultures contaminated with *Aspergillus niger*.^{302, 303} The latter report mentioned no use of barrier containment and noted that the HEPA filtration system was overloaded with dust. An outbreak of pseudobacteremia due to *Bacillus* spp. occurred during hospital construction over a storage area for blood culture bottles.²⁰³ Airborne spread of *Bacillus* spp. spores resulted in contamination of the bottles' plastic lids, which were not disinfected or handled with proper aseptic technique prior to collection of blood samples.

5. Environmental Infection Control Measures for Special Healthcare Settings

Areas in healthcare facilities that require special ventilation include: 1) operating rooms; 2) PE rooms used by high-risk, immunocompromised patients; and 3) AII rooms for isolation of patients with airborne infections due to *M. tuberculosis*, VZV, or measles virus. The number of rooms required for PE and AII are determined by a risk assessment of the healthcare facility.⁶

a. Protective Environments (PE)

Although the exact configuration and specifications of PE might differ among hospitals, these care areas for high-risk, immunocompromised patients are designed to minimize fungal spore counts in air by maintaining: 1) filtration of incoming air by using central or point-of-use HEPA filters; 2) directed room airflow [i.e., from supply on one side of the room, across the patient, and out through the exhaust on the opposite side of the room]; 3) positive room air pressure relative to the corridor [supplied to the room at a rate that is 150 ft³/min. greater than the rate of exhaust]; 4) well-sealed rooms; and 5) ≥ 12 ACH.^{44, 244, 247, 304 - 307} Directed room airflow in PE rooms is not laminar airflow, as parallel air streams are not generated. Studies attempting to demonstrate patient benefit from laminar airflow in a PE setting are equivocal.^{304, 306 - 313}

Airflow direction at the entrances to these areas should be maintained and verified, preferably on a daily basis, using either a visual means of indication (e.g., smoke tubes) or manometers. Facility service structures can interfere with the proper unidirectional airflow from the patients' rooms to the adjacent corridor. In one study, *Aspergillus* spp. infections in a critical-care unit may have been associated with a pneumatic specimen transport system, a linen disposal duct system, and central vacuum lines for housekeeping, all of which disrupted proper airflow from the patients' rooms to the outside and allowed entry of fungal spores into the unit.³¹⁴

The use of surface fungicide treatments is becoming more common, especially for building materials.³¹⁵ Copper-based compounds have demonstrated anti-fungal activity and are often applied to wood or paint. Copper-8-quinolinolate was used on environmental surfaces contaminated with *Aspergillus* spp. to control one reported outbreak of aspergillosis.²⁹⁹ The compound was also incorporated into the fireproofing material of a newly constructed hospital to help decrease the

environmental spore burden.³⁰⁴

The use of a NIOSH-approved respirator by high-risk, immunocompromised patients may be considered as part of the strategy to protect such patients when they leave PE for treatments elsewhere in the facility.²⁷² These personal protective devices have been shown under experimental conditions to be effective in preventing both the inhalation of respirable particles and the reaerosolization of exhaled particles.^{316 - 320} In order to prevent reaerosolization of exhaled particles, filtering facepieces (disposable respirators) without exhalation valves must be used. Under simulated breathing conditions, N95 respirators collected $\geq 95\%$ of particles with diameters ranging from 0.1 - 0.3 μm and achieved filtering efficiencies of $\geq 99.5\%$ with particles of 0.75 μm diameter.^{318, 319} Reaerosolization rates of $\leq 0.1\%$ were noted under test conditions simulating violent coughing or sneezing.³¹⁹ The clinical efficacy of these devices in preventing aspergillosis and other opportunistic fungal infections has not been fully evaluated.

b. Airborne Infection Isolation Areas (AII)

All acute-care inpatient facilities need at least one room equipped to house patients with airborne infection (AII). Guidelines for the prevention of healthcare-acquired TB have been published in response to multiple reports of healthcare-associated transmission of multiresistant strains.^{4, 321} In reports documenting healthcare-acquired TB, investigators have noted a failure to comply fully with prevention measures in established guidelines.^{322 - 336} These gaps highlight the importance of prompt recognition of the disease, isolation of patients, proper treatment, and engineering controls.

Salient features of engineering controls for AII areas include: 1) use of negative pressure rooms with close monitoring of airflow direction using manometers or visual indicators (e.g., smoke tubes) placed in the room with the door closed; 2) ≥ 12 ACH for area renovation or new construction; and 3) air from negative pressure rooms and treatment rooms exhausted directly to the outside if possible.^{4, 120} When the recirculation of air from these rooms is unavoidable, HEPA filters should be installed in the exhaust duct leading from the room to the general ventilation system. In addition to UVGI fixtures in the room, UVGI can be placed in the ducts as an adjunct measure to HEPA filtration, but cannot replace the HEPA filter.^{4, 337}

Cough-inducing procedures such as endotracheal intubation and suctioning, diagnostic sputum induction, aerosol treatments, and bronchoscopy require similar precautions. Sputum induction performed in an area without whole room negative pressure (e.g., clinic, emergency department) requires an enclosed booth with these specifications: 1) 12 ACH; 2) negative pressure; 3) an exhaust rate at least 50 ft^3/min . directly to the outside; and 4) an air volume differential of $\geq 100 \text{ft}^3/\text{min}$.^{338, 339} A HEPA filter is required in the exhaust grille if the air is recirculated or exhausted to areas near air-intake vents, persons, or animals. If such a booth or enclosed space is unavailable, then adequate time must be allowed for sufficient ACH needed to remove 99.9% of airborne particles (Appendix B).⁴

A special case is the management of patients requiring PE (i.e., allogeneic HSCT patients) who concurrently have TB or other airborne infection. For this type of patient treatment, an anteroom is required as per AIA guidelines; the pressure differential of an anteroom can be positive or negative relative to the patient in the room.¹²⁰ An anteroom can act as an airlock. If the anteroom is positive relative to the air space in the patient's room, staff members do not have to mask prior to entry into the anteroom as long as there is direct air exhaust to the outside and a minimum of 10 ACH.¹²⁰ If an anteroom is not available, use of a portable, industrial grade HEPA filter unit may help to increase the number of ACHs, but there must be a fresh air source to achieve the proper air exchange rate. Incoming ambient air should receive HEPA filtration.

Figure 2. Positive-Pressure Room^a

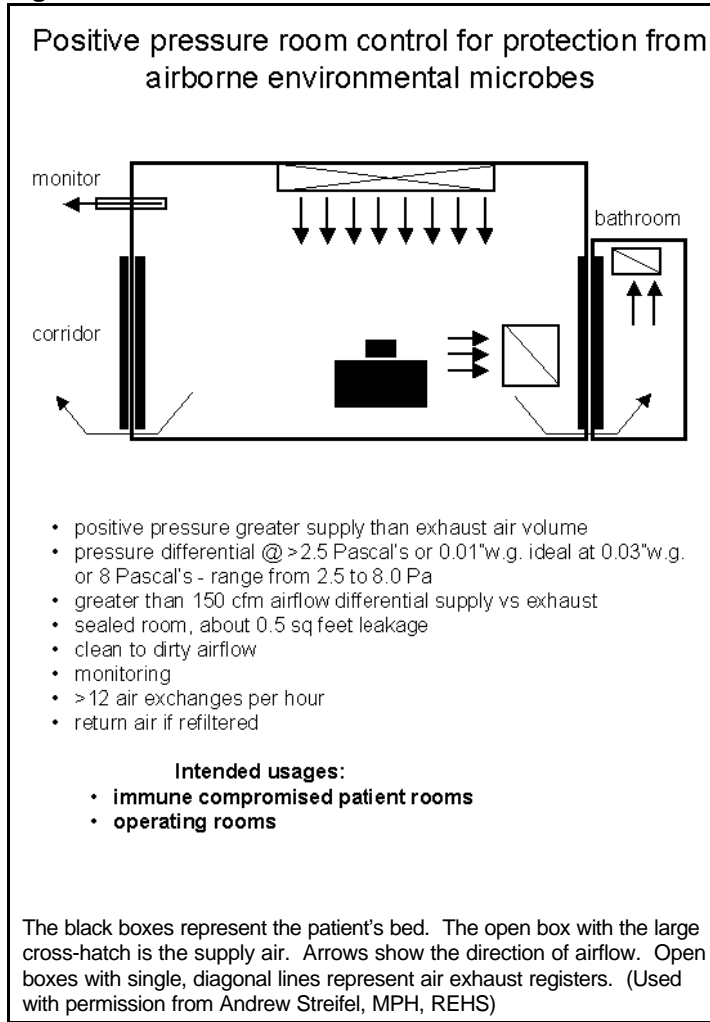


Figure 3. Negative-Pressure Room^a

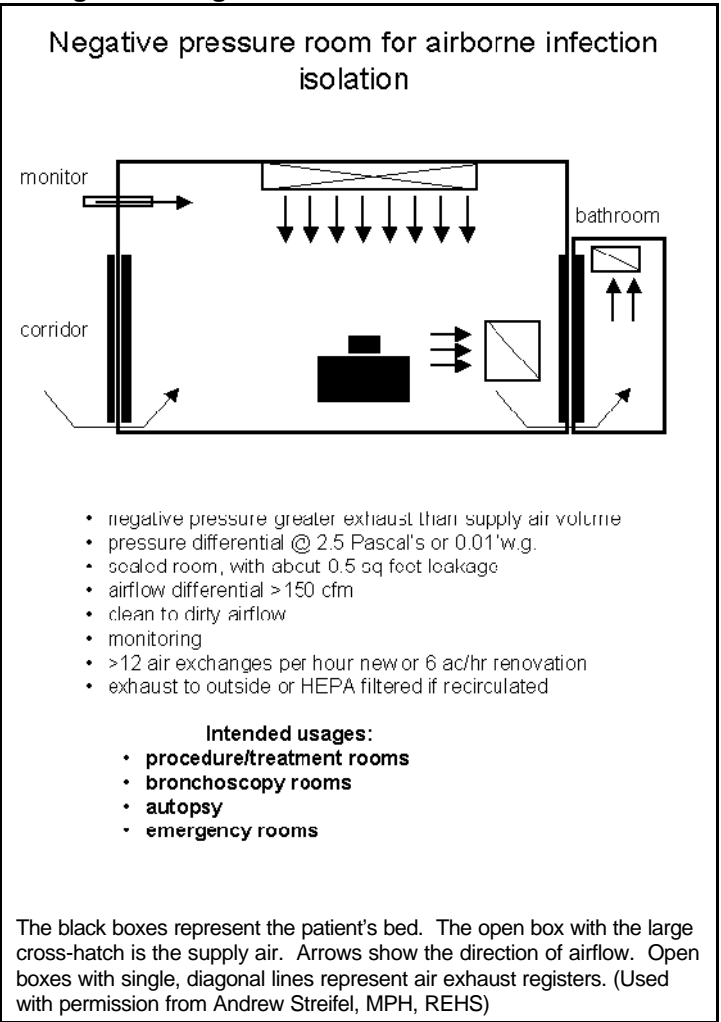
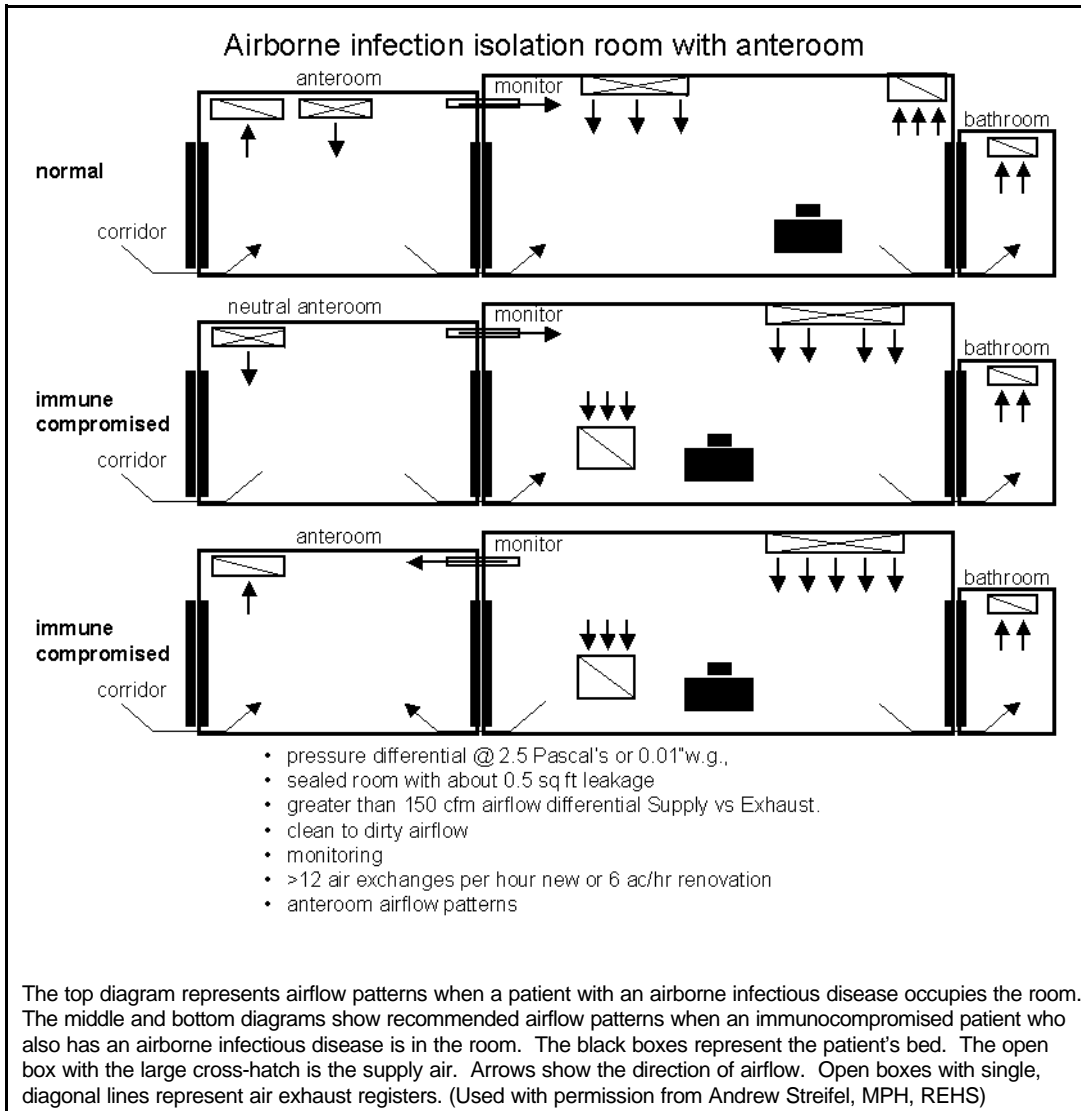


Figure 4. Airborne Infection Isolation Room With Anteroom^a



c. Operating Rooms

Operating room air may contain microorganisms, dust, aerosol, lint, skin squamous epithelial cells, and respiratory droplets. The microbial level in operating room air is directly proportional to the number of people moving about in the room.³⁴⁰ One study documented lower infection rates with coagulase-negative staphylococci among patients when operating room traffic during the procedure was limited.³⁴¹ Therefore, efforts should be made to minimize personnel traffic during operations. Outbreaks of surgical site infections (SSIs) caused by group A beta-hemolytic streptococci have been traced to airborne transmission from colonized operating room personnel to patients.^{148 - 152} Several potential healthcare-associated pathogens (e.g., *Staphylococcus aureus* and *Staphylococcus epidermidis*) and drug-resistant organisms have also been recovered from areas adjacent to the surgical field,³⁴² but the extent to which the presence of bacteria near the surgical field influences the development of postoperative SSIs is not clear.³⁴³

Operating rooms should be maintained at positive pressure with respect to corridors and adjacent areas.³⁴⁴ Operating rooms should not have variable air handling systems. Conventional operating room ventilation systems produce a minimum of about 15 ACH of filtered air for thermal control, three (20%) of which must be fresh air.^{120, 345} Air should be introduced at the ceiling and exhausted near the floor.^{345, 346}

Laminar airflow and UVGI have been suggested as adjunct measures to reduce SSI risk for certain operations. Laminar

airflow is designed to move particle-free air over the aseptic operating field at a uniform velocity (0.3 to 0.5 µm/sec), sweeping away particles in its path. This air flow can be directed vertically or horizontally, and recirculated air is passed through a HEPA filter.^{347 - 350} Neither laminar airflow nor UV light, however, has been conclusively shown to decrease overall SSI risk.^{344, 351 - 357}

The use of general anesthesia in TB patients poses special infection control challenges because intubation can induce coughing. Although operating room suites at 15 ACH exceed the air exchanges required for TB isolation, the positive airflow relative to the corridor could result in healthcare-associated transmission of TB to operating room personnel. The AIA currently does not recommend changing pressure from positive to negative or setting it to neutral, and it is doubtful that many facilities have the capability to do so.¹²⁰ Elective surgery on infectious TB patients should be postponed until they have received adequate drug therapy. When emergency surgery is indicated for an infectious TB patient, infection control measures as outlined in Table 14 appear to be reasonable.

Table 14. Strategy for Managing TB Patients and Preventing Airborne Transmission in Operating Rooms^{4, 358}

-
- 1) If emergency surgery is indicated on a patient with active TB, schedule the TB patient as the last surgical case to provide maximum time for air cleaning (ACHs).
 - 2) Operating room personnel should use NIOSH-approved N95 respirators.
 - 3) Keep the door closed after the patient is intubated.
 - 4) Allow adequate time for sufficient ACH necessary to remove 99.9% of airborne particles (Appendix B):
 - a) after the patient is intubated and particularly if intubation induces coughing;
 - b) if the door to the operating suite must be opened; and intubation induces coughing in the patient; or
 - c) after the patient is extubated and suctioned (unless there is a closed suctioning system).
 - 5) Extubate the patient in the operating room or allow the patient to recover in AII rather than in the regular open recovery facilities.
 - 6) Temporary use of a portable, industrial grade HEPA filter may expedite removal of airborne contaminants (fresh air exchange requirements for proper ventilation must still be met).*
 - 7) Breathing circuit filters (e.g., Pall BB 25A, BB 100, and HME 15-22) with removal efficiencies of >99.99% to >99.999% for *Mycobacterium bovis* (a surrogate for *Mycobacterium tuberculosis*) at a minimum total concentration of 10⁴ CFU can be used as an adjunct infection control measure.³⁵⁹
-

* Portable HEPA filter units previously used in construction areas should not be used in subsequent patient care.

Table 15. Summary of Ventilation Specifications in Selected Areas of Healthcare Facilities³⁵

Specifications	Airborne Isolation (AII) ^a	Protective Environment (PE)	Critical Care Room ^b	Isolation Anteroom	Operating Room
Air pressure ^c	Negative	Positive	Positive, negative, or neutral	Positive or negative	Positive
Room air changes	≥12 ACH (for renovation or new construction)	>12 ACH	≥6 ACH	≥10 ACH	15 or 25 ACH
Sealed ^d	YES	YES	NO	YES	YES
Filtration supply	90% (dust-spot ASHRAE 52-76)	99.97% ^e	≥ 90%	≥ 90%	90%
Recirculation	NO ^f	YES	YES	NO	YES

a. Includes bronchoscopy suites.

b. Positive pressure and HEPA filters may be preferred in some rooms in intensive care units (ICUs) caring for large numbers of immunocompromised patients.

c. Clean-to-dirty: negative to infectious patient, positive away from a compromised patient.

d. Minimized infiltration for ventilation control. Pertains to windows, closed doors, surface joints.

e. Fungal spore filter at point of use - HEPA at 99.97% of 0.3 μm particles.

f. Recirculated air may be used if the exhaust air is first processed through a HEPA filter.

g. Used with permission of the publisher (reference 35).

6. Other Aerosol Hazards in Healthcare Facilities

In addition to infectious bioaerosols, there are several important non-infectious indoor air quality issues for healthcare facilities. The presence of sensitizing and allergenic agents and irritants in the workplace is increasing in importance. Some common agents include ethylene oxide, glutaraldehyde, formaldehyde, hexachlorophene, and latex allergens.³⁶⁰ Asthma and dermatologic and systemic reactions often result with exposure to these chemicals. Anesthetic gases and aerosolized medications such as ribavirin, pentamidine, and aminoglycosides represent some of the emerging potentially hazardous exposures to healthcare workers. Containment of the aerosol at the source is the first level of engineering control, but personal protective equipment (e.g., masks, respirators, glove liners) to distance the worker from the hazard may be needed as well.

Laser plumes or surgical smoke represent another potential risk for healthcare workers.^{361 - 363} Lasers transfer electromagnetic energy into tissues, resulting in the release of a heated plume that includes particles, gases, tissue debris, and offensive smells. One concern is that aerosolized infectious material in the laser plume might reach the nasal mucosa of surgeons and adjacent personnel. Although some viruses (i.e., varicella-zoster virus, pseudorabies virus, herpes simplex virus) apparently do not aerosolize efficiently,^{364, 365} other viruses and a variety of bacteria (e.g., human papilloma virus [HPV], HIV, coagulase-negative *Staphylococcus*, *Corynebacterium* spp., *Neisseria* spp.) have been detected in laser plumes.^{366 - 372} The presence of an infectious agent in a laser plume may not, however, be sufficient to cause disease from airborne exposure, especially if the normal mode of transmission for the agent is not airborne. There is no evidence that HIV or hepatitis B virus (HBV) has been transmitted via aerosolization and inhalation.³⁷³

Although continuing studies are needed to fully evaluate the risk of laser plumes to the surgical team, it is prudent to follow NIOSH recommendations³⁶³ and the *Recommended Practices for Laser Safety in Practice Settings* developed by the Association of periOperating Room Nurses (AORN).³⁷⁴ These practices include the use of: 1) high-filtration surgical masks and possibly full face shields;³⁷⁵ 2) central wall suction units with in-line filters to collect particulate matter from

minimal plumes; and 3) dedicated mechanical smoke exhaust systems with a high-efficiency filter to remove large amounts of laser plume. Use of a smoke evacuator or needle aspirator can help protect healthcare workers when excising and draining a TB abscess.⁴

D. Water

1. Modes of Transmission of Waterborne Diseases

Moist environments and aqueous solutions in healthcare settings have the potential to serve as reservoirs for waterborne microorganisms. Under favorable environmental circumstances (e.g., temperature, source of nutrition), these microorganisms can proliferate to great numbers or may also remain for long periods in highly stable, environmentally-resistant yet infectious forms. Modes of transmission for waterborne infections include: 1) direct contact, such as during hydrotherapy; 2) ingestion of water, such as from consuming contaminated ice; 3) indirect-contact transmission, such as from an improperly reprocessed medical device;⁶ 4) inhalation of aerosols dispersed from water sources;³ and 5) aspiration of contaminated water. The first three modes of transmission are commonly associated with infections due to gram-negative bacteria and non-tuberculous mycobacteria (NTM). Aerosols generated from water sources contaminated with *Legionella* spp. often serve as the vehicle for introducing these pathogens to the respiratory tract.³⁷⁶

2. Waterborne Infectious Diseases in Healthcare Facilities

a. Legionellosis

Legionellosis is a collective term describing infection produced by *Legionella* spp., whereas Legionnaires' disease is a multisystem illness with pneumonia.³⁷⁷ The clinical and epidemiologic aspects of these diseases, summarized in Table 16, are discussed extensively in another guideline.³ Although Legionnaires' disease is a respiratory infection, infection control measures intended to prevent healthcare-associated cases center on water quality since the principal reservoir for *Legionella* spp. is water.

Legionella spp. are commonly found in various natural and manmade aquatic environments^{378, 379} and can enter healthcare facility water systems in low or undetectable numbers.^{380, 381} Cooling towers, evaporative condensers, heated potable water distribution systems, and locally produced distilled water can provide environments for multiplication of legionellae.^{382 - 386} In several hospital outbreaks, patients were considered to be infected through exposure to contaminated aerosols generated by cooling towers, showers, faucets, respiratory therapy equipment, and room-air humidifiers.^{387 - 396} Factors that enhance colonization and amplification of legionellae in manmade water environments include: 1) temperatures of 25° - 42°C [77°F - 107.6°F];^{397 - 401} 2) stagnation;⁴⁰² 3) scale and sediment;⁴⁰³ and 4) presence of certain free-living aquatic amoebae that can support intracellular growth of legionellae.^{403, 404} The bacteria multiply within single-cell protozoa in the environment and within alveolar macrophages in humans.

Table 16. Clinical and Epidemiologic Characteristics of Legionellosis/Legionnaires' Disease

		References
Causative Agent	<i>Legionella pneumophila</i> (90% of infections); <i>L. micdadei</i> , <i>L. bozemanii</i> , <i>L. dumoffii</i> , <i>L. longbeachii</i> , (14 additional species can cause infection in humans)	377, 405 - 408
Mode of Transmission	Aspiration of water, direct inhalation of water aerosols	3, 376, 377, 405, 407, 409
Infection Associated With:	Exposure to environmental sources of <i>Legionella</i> spp. (i.e., water or water aerosols)	31, 33, 387 - 396, 410 - 413
Clinical Syndromes and Diseases	Two distinct illnesses: Pontiac fever - a milder influenza-like illness; severe, progressive Pneumonia that may be associated with cardiac, renal, and gastrointestinal involvement.	3, 406 - 408, 414 - 421
Patient Populations at Greatest Risk	Immunosuppressed patients (e.g., transplant patients, cancer patients, patients receiving corticosteroid therapy); immunocompromised patients (e.g., surgical patients, patients with underlying chronic lung disease, dialysis patients); elderly persons, patients who smoke	377, 405, 406, 422 - 432
Occurrence	Proportion of community-acquired pneumonia due to <i>Legionella</i> spp. ranges from 1% - 5%; estimated annual incidence among the general population 8,000 - 18,000 cases in the U.S.; incidence of healthcare-associated pneumonia (0% - 14%) may be underestimated if appropriate laboratory diagnostic methods are unavailable.	405, 406, 433 - 443
Mortality Rate	5% - 30% of patients in outbreaks; rate is higher among healthcare-associated pneumonia cases compared to that for community-acquired pneumonia patients	377, 405, 406

b. Other Gram-Negative Bacterial Infections

Other gram-negative bacteria present in finished or potable water can also cause healthcare-associated infections. Clinically important organisms in tap water include *Pseudomonas aeruginosa*, *Pseudomonas* spp., *Burkholderia cepacia*, *Ralstonia pickettii*, *Stenotrophomonas maltophilia*, and *Sphingomonas* spp. (Tables 17 and 18). These organisms are largely opportunistic; immunocompromised patients are at greatest risk of developing infection. Medical conditions associated with these bacterial agents range from colonization of the respiratory and urinary tracts to deep, disseminated infections that can result in pneumonia and bloodstream bacteremia. Colonization by any of these organisms often precedes the development of infection. The use of tap water in medical care (e.g., in direct patient care, as a diluent for solutions, as a water source for medical instruments and equipment, during the final stages of instrument disinfection), therefore presents a potential risk of exposure. Colonized patients can also serve as a source of contamination, particularly for moist environments of medical equipment (e.g., ventilators).

In addition to *Legionella* spp., *Pseudomonas aeruginosa* and *Pseudomonas* spp. are among the most important of the clinically-relevant, gram-negative, healthcare-associated pathogens identified from water. *Pseudomonas* spp., along with other gram-negative, non-fermentative bacteria, have minimal nutritional requirements (i.e., these organisms can grow in distilled water) and can tolerate a variety of physical conditions (e.g., temperature fluctuations); these attributes are important for these organisms' success as healthcare-associated pathogens and widespread distribution in moist environments. Measures to prevent the spread of these organisms and other waterborne gram-negative bacteria include handwashing, use of gloves and other barrier precautions; and eliminating potentially contaminated environmental reservoirs.^{444, 445}

Table 17. *Pseudomonas aeruginosa* Infections in Healthcare Facilities

		References
Clinical syndromes	Septicemia; pneumonia (particularly ventilator-associated); chronic respiratory infections among cystic fibrosis patients; urinary tract infections; skin and soft-tissue infections (tissue necrosis and hemorrhage); burn wound infections; folliculitis; endocarditis; central nervous system infections (meningitis, abscess); eye infections; bone and joint infections	446 - 483
Modes of transmission	Direct contact with water, aerosols, aspiration, indirect transfer from moist environmental surfaces via healthcare worker hands	28, 482 - 486
Environmental sources of Pseudomonads in healthcare settings	Potable (tap) water; distilled water; antiseptic solutions; sinks, hydrotherapy pools, whirlpools; water baths; lithotripsy therapy; dialysis water; eyewash stations; flower vases	28, 29, 446, 448, 487 - 498
Environmental sources of Pseudomonads in the community	Fomites (e.g., drug injection equipment) stored in contaminated water	474, 475
Patient populations at greatest risk	Intensive care unit patients (including NICU), transplant patients (organ and hematopoietic stem cell), neutropenic patients, burn therapy and hydrotherapy patients, patients with malignancies, cystic fibrosis patients, patients with underlying medical conditions, dialysis patients	28, 446, 447, 452, 457, 473, 486 - 488, 491, 492, 499 - 504

Table 18. Other Gram-Negative Bacteria Associated with Water and Moist Environments

Implicated Environmental Vehicle	References
<i>Burkholderia cepacia</i>	
Distilled water	505
Contaminated solutions and disinfectants	506, 507
Dialysis machines	505
Nebulizers	508 - 510
Water baths	511
Intrinsically-contaminated mouthwash	512
Ventilator temperature probes	513
<i>Stenotrophomonas maltophilia, Sphingomonas spp.</i>	
Distilled water	514, 515
Contaminated solutions and disinfectants	507
Dialysis machines	505
Nebulizers	508 - 510
Water	516
Ventilator temperature probes	517
<i>Ralstonia pickettii</i>	
Fentanyl solutions	518
Chlorhexidine	519
Distilled water	519
Contaminated respiratory therapy solution	519, 520
<i>Serratia marcescens</i>	
Potable water	521
Contaminated antiseptics - benzalkonium chloride, chlorhexidine	522 - 524
Contaminated disinfectants - quaternary ammonium compounds	525, 526
Contaminated disinfectants - glutaraldehyde	525, 526
<i>Acinetobacter spp.</i>	
Medical equipment that collects moisture - mechanical ventilators, cool mist humidifiers, vaporizers, mist tents	527 - 534
Room humidifiers	531, 533
Environmental surfaces	535 - 542
<i>Enterobacter spp.</i>	
Humidifier water	543
Intravenous fluids	544 - 556
Unsterilized cotton swabs	551
Ventilators	543, 547
Rubber piping on a suctioning machine	543, 547
Blood gas analyzers	548

Two additional gram-negative bacterial pathogens which can proliferate in moist environments are *Acinetobacter* spp. and *Enterobacter* spp.^{549, 550} Members of both genera are responsible for healthcare-associated episodes of colonization, bloodstream infections, pneumonia, and urinary tract infections among medically-compromised patients, especially those in intensive care units and burn therapy units.^{544, 550 - 561} Infections due to *Acinetobacter* spp. represent a significant clinical problem. Average infection rates are higher during July - October compared to rates noted from November - June.⁵⁶² Mortality associated with *Acinetobacter* bacteremia ranges from 17% - 52%, and rates as high as 71% have been reported for pneumonia due to infection with either *Acinetobacter* spp. or *Pseudomonas* spp.^{552 - 554} Multi-drug resistance, especially concerning third generation cephalosporins for *Enterobacter* spp., contributes to increased

morbidity and mortality.^{547, 550}

Patients and healthcare workers represent important sources of either *Acinetobacter* spp. or *Enterobacter* spp., contributing to environmental contamination of surfaces and equipment, especially in intensive care areas because of the nature of the medical equipment (e.g., ventilators) and the moisture associated with this equipment.^{527, 549, 550, 563} Hand carriage and hand transfer are important factors for healthcare-associated transmission of these organisms, and for *Serratia marcescens*.⁵⁶⁴ *Enterobacter* spp. are primarily spread in this manner among patients by the hands of healthcare workers.^{545, 565} *Acinetobacter* spp. have been isolated from the hands of 4% to 33% of healthcare workers in some studies,^{563 - 568} and transfer of an epidemic strain of *Acinetobacter* from patients' skin to healthcare workers' hands has been demonstrated experimentally.⁵⁶⁹ *Acinetobacter* infections and outbreaks have been attributed to hand transfer of the organisms and to contaminated medical equipment and materials, especially devices that collect moisture (e.g., ventilators, cool mist humidifiers, vaporizers, mist tents) or have other contact with water of uncertain quality (e.g., rinsing a ventilator circuit in tap water).^{527 - 534} Strict adherence to hand hygiene or handwashing helps prevent the spread of both *Acinetobacter* spp. and *Enterobacter* spp.^{555, 570}

Acinetobacter spp. have also been detected on a variety of dry environmental surfaces (e.g., bed rails, counters, sinks, bed cupboards, bedding, floors, telephones, medical charts) in the vicinity of colonized or infected patients.^{535 - 542} In two studies, the survival periods of *A. baumannii* and *A. calcoaceticus* on dry surfaces approximated that for *Staphylococcus aureus* (e.g., 26 - 27 days).^{571, 572} Because *Acinetobacter* spp. may come from numerous sources at any given time, laboratory investigation of healthcare-associated *Acinetobacter* infections may involve techniques to determine biotype, antibiotype, plasmid profile, and genomic fingerprinting (macrorestriction analysis) to accurately identify sources and modes of transmission of the organism(s).⁵⁷³

c. Infections and Pseudoinfections Due to Non-Tuberculous Mycobacteria

Non-tuberculous mycobacteria spp. (NTM) are acid-fast bacilli (AFB) commonly found in potable water. NTM include both saprophytic and opportunistic organisms. Many NTM are of low pathogenicity, and some measure of host impairment is necessary to enhance clinical disease.⁵⁷⁴ The four most common forms of human disease associated with NTM are: 1) pulmonary disease in adults; 2) cervical lymph node disease in children; 3) skin, soft tissue, and bone infections; and 4) disseminated disease in immunocompromised patients.^{574, 575} Person-to-person transmission of NTM infection does not appear to occur, and close contacts of patients are not readily infected, even though a patient may be shedding large numbers of organisms.^{574, 576 - 578} NTM are spread via all the modes of transmission associated with water. In addition to healthcare-associated outbreaks of clinical disease, NTM can colonize patients in healthcare facilities through consumption of contaminated water or ice, or inhalation of aerosols.^{579 - 583} Colonization following NTM exposure occurs when a patient's local defense mechanisms are impaired; overt clinical disease is usually not described.⁵⁸⁴ Patients may have positive sputum cultures in the absence of clinical disease.

Table 19. Non-Tuberculous Mycobacteria - Environmental Vehicles

Vehicles Associated with Infections or Colonization	References
<i>M. abscessus</i>	
Inadequately sterilized medical instruments	585
<i>M. avium complex (MAC)</i>	
Potable water	586 - 588
<i>M. chelonae</i>	
Dialysis, reprocessed dialyzers	31, 32
Inadequately sterilized medical instruments, jet injectors	589, 590
Contaminated solutions	591, 592
Hydrotherapy tanks	593
<i>M. fortuitum</i>	
Aerosols from showers or other sources	583, 584
Ice	580
Inadequately sterilized medical instruments	579
Hydrotherapy tanks	594
<i>M. marinum</i>	
Hydrotherapy tanks	595
<i>M. ulcerans</i>	
Potable water	596
Vehicles Associated with Pseudo-outbreaks	
<i>M. chelonae</i>	
Potable water used during bronchoscopy and instrument reprocessing	597
<i>M. fortuitum</i>	
Ice	598
<i>M. gordonae</i>	
Deionized water	599
Ice	581
Laboratory solution (intrinsicly contaminated)	600
<i>M. kansasii</i>	
Potable water	601
<i>M. terrae</i>	
Potable water	602
<i>M. xenopi</i>	
Potable water	601, 603, 604

Using tap water during patient procedures, specimen collection and transport, or in the final steps of instrument reprocessing can result in pseudo-outbreaks of NTM contamination.^{598, 602, 603} NTM pseudo-outbreaks of *M. chelonae*, *M. gordonae*, and *M. xenopi* have been associated with both bronchoscopy and gastrointestinal endoscopy when tap water is used to provide irrigation to the site or to rinse off the viewing tip *in situ*, or if the instruments are inappropriately reprocessed with tap water in the final steps.^{597, 599, 604}

NTM can be isolated from both natural and manmade environments. Numerous studies have identified various NTM in municipal water systems and in hospital water systems and storage tanks.^{587, 588, 596, 601, 605 - 609} Some NTM species (e.g., *M. xenopi*) can survive in water at 45°C (113°F), and can be isolated from hot water taps, which can pose a problem for hospitals that lower the temperature of their hot water systems.⁶⁰¹ Other NTM (e.g., *M. kansasii*, *M. gordonae*, *M. fortuitum*, and *M. chelonae*) cannot tolerate high temperatures and are found associated more often with cold water lines and taps.⁶⁰⁶

NTM have a high degree of resistance to chlorine; they can tolerate free chlorine concentrations of 0.05 - 0.2 mg/L (0.05 - 0.2 ppm) found at the tap.^{576, 610, 611} They are 20 - 100 times more resistant to chlorine compared to coliforms, and slow-

growing strains of NTM appear to be more resistant to chlorine inactivation compared to fast-growing NTM.⁶¹² Slow-growing NTM species (e.g., *M. avium*, *M. kanasii*) have also demonstrated some resistance to formaldehyde and glutaraldehyde, which has posed problems for reuse of hemodialyzers.³¹ The ability of NTM to form biofilms at fluid-surface interfaces (e.g., interior surfaces of water pipes) contributes to the organisms' resistance to chemical inactivation and provides a microenvironment for growth and proliferation.^{613, 614}

d. Cryptosporidiosis

Cryptosporidium parvum is a protozoan parasite that causes self-limiting gastroenteritis in normal hosts but can cause severe, life-threatening disease in immunocompromised patients. First recognized as a human pathogen in 1976, *C. parvum* can be present in natural and finished waters after fecal contamination from either human or animal sources.^{615 - 618}

The health risks associated with drinking potable water contaminated with small numbers of *C. parvum* oocysts are unknown.⁶¹⁹ It remains to be determined if immunosuppressed persons are more susceptible to lower doses of oocysts than are immunocompetent persons, or if strains of *C. parvum* vary in their infectious dose and their ability to cause disease. One study demonstrated that a median 50% infectious dose (ID₅₀) of 132 oocysts was sufficient to cause infection among healthy volunteers.⁶²⁰ In a small study population of 17 healthy adults with pre-existing antibody to *C. parvum*, the ID₅₀ was determined to be 1,880 oocysts, more than 20-fold higher than in seronegative persons.⁶²¹ These data suggest that pre-existing immunity derived from previous exposures to *Cryptosporidium* offers some protection from infection and illness that ordinarily would result from exposure to low numbers of oocysts.^{621, 622}

Oocysts, particularly those with thick walls, are environmentally resistant, but their survival in water is poorly understood.⁶¹⁸ The prevalence of *Cryptosporidium* in the United States drinking water supply is, however, notable. Two surveys of approximately 300 surface water supplies revealed that 55% - 77% of the water samples contained *Cryptosporidium* oocysts.^{623, 624} Because the oocysts are highly resistant to common disinfectants (e.g., chlorine) used to treat drinking water, filtration of the water is important in reducing the risk of waterborne transmission. Coagulation-flocculation and sedimentation, when used with filtration, can collectively achieve approximately a 2.5 log₁₀ reduction in the number of oocysts.⁶²⁵ However, outbreaks have been associated with both filtered and unfiltered drinking water systems (e.g., the 1993 outbreak in Milwaukee, Wisconsin, that affected 400,000 people).^{618, 626 - 628} The presence of oocysts in the water is not an absolute indicator that infection will occur when the water is consumed, nor does the absence of detectable oocysts guarantee that infection will not happen. Healthcare-associated outbreaks of cryptosporidiosis have been described primarily among groups of elderly patients and immunocompromised persons.⁶²⁹

3. Water Systems in Healthcare Facilities

a. Basic Components and Point-of-Use Fixtures

Treated municipal water comes into a healthcare facility via the water mains and is distributed throughout the building(s) by a network of pipes constructed of galvanized iron, copper, and polyvinylchloride (PVC). The pipe runs should be as short as practical. Where recirculation is employed, the pipe runs should be insulated and long, dead legs avoided in efforts to minimize the potential for water stagnation to occur which favors the proliferation of *Legionella* and NTM in the system. In high-risk applications insulated recirculation loops should be incorporated as a design feature.

Each water service main, branch main, riser, and branch (to a group of fixtures) has a valve and a means to reach the valves via an access panel.¹²⁰ Each fixture has a stop valve. Valves permit the isolation of a portion of the water system within a facility during repairs or maintenance. Vacuum breakers in the lines prevent water from back-flowing into the system.

Healthcare facilities generate hot water from mains water using a boiler system. Hot water heaters and storage vessels for such systems should have a drainage facility at the lowest point and the heating element should be located as close as possible to the bottom of the vessel to facilitate mixing and prevent water temperature stratification. Those hot or cold water systems which incorporate an elevated holding tank should be inspected and cleaned annually. Lids should fit closely to exclude foreign materials.

The most common point-of-use fixtures for water in patient-care areas are sinks, faucets, aerators, showers, and toilets;

eye-wash stations are found primarily in laboratories. The potential for these fixtures to serve as a reservoir for pathogenic microorganisms has long been recognized (Table 20).^{489, 630 - 632} Wet surfaces and the production of aerosols facilitate the multiplication of and dispersion of microbes. The level of risk associated with aerosol production from point-of-use fixtures varies. Aerosols from shower heads and aerators have been linked to a limited number of clusters of gram-negative bacterial colonizations and infections, including Legionnaires' disease, especially in areas where immunocompromised patients are present (e.g., surgical intensive care units, transplant units, and oncology units).^{393, 396, 632 - 635} In one report, clinical infection was not evident among immunocompetent persons (e.g., hospital staff) who used hospital showers when *L. pneumophila* was present in the water system.⁶³⁶ Given the infrequency of reported outbreaks associated with faucet aerators, expert opinion on the removal of these devices from general use is mixed. If additional clusters of infections or colonizations occur in high-risk patient-care areas, then it may be prudent to clean and decontaminate the aerators or remove them.^{634, 635} ASHRAE recommends cleaning and monthly disinfection of aerators in high-risk patient-care areas as part of *Legionella* control measures.⁶³⁷ Although aerosols are produced with toilet flushing,^{638, 639} there is no epidemiologic evidence to suggest that these aerosols pose a direct infection hazard.

Although not considered a standard point-of-use fixture, decorative fountains are increasingly being installed in healthcare facilities and other public buildings. Aerosols from a decorative fountain in a hotel lobby transmitted *L. pneumophila* serogroup 1 infection to a small cluster of older adults.⁶⁴⁰ The fountain had been irregularly maintained, and water in the fountain may have been heated by submersed lighting, all of which favored the proliferation of *Legionella* in the system.⁶⁴⁰ Because of the potential for generations of infectious aerosols, a prudent prevention measure is to avoid locating these fixtures in or near high-risk patient-care areas and to adhere to written policies for routine maintenance of fountains that are installed.

Table 20. Water and Point-of-Use Fixtures as Sources and Reservoirs of Waterborne Pathogens^a

Reservoir	Associated Pathogen(s)	Transmission	Strength of Evidence ^b	Prevention and Control	References
Potable water	<i>Pseudomonas</i> , NTM	Contact	Moderate	Follow public health guidelines.	(See Tables 17 - 19)
Potable water	<i>Legionella</i>	Aerosol inhalation	Moderate	Provide supplemental treatment for water.	(See Table 16)
Holy water	Gram-negative bacteria	Contact	Low	Avoid contact with severe burn injuries. Minimize use among immunocompromised patients.	641
Dialysis water	Gram-negative bacteria	Contact	Moderate	Dialysate should be ≤ 2000 CFU/mL; Water should be ≤ 200 CFU/mL.	2, 505, 642 - 644
Water baths	<i>Pseudomonas</i> , <i>Burkholderia</i> , <i>Acinetobacter</i>	Contact	Moderate	Add germicide to the water; wrap transfusion products in protective plastic wrap if using the bath to modulate the temperature of these products.	29, 511, 645, 646
Tub immersion	<i>Pseudomonas</i> , <i>Enterobacter</i> , <i>Acinetobacter</i>	Contact	Moderate	Drain and disinfect tub after each use; consider adding germicide to the water; water in large hydrotherapy pools should be properly disinfected and filtered.	647 - 652
Ice and ice machines	NTM, <i>Enterobacter</i> , <i>Pseudomonas</i> , <i>Cryptosporidium</i> , <i>Legionella</i>	Ingestion, contact	Moderate Low	Clean periodically; use automatic dispenser (avoid open chest storage compartments in patient areas).	579, 653 - 656
Faucet aerators	<i>Legionella</i>	Aerosol inhalation	Moderate	Clean and disinfect monthly; consider removing if additional infections occur.	396, 637
Faucet aerators	<i>Pseudomonas</i> , <i>Acinetobacter</i> , <i>Stenotrophomonas</i>	Contact, droplet	Low	No precautions are necessary at present in immunocompetent patient care areas.	634, 635, 657
Sinks	<i>Pseudomonas</i>	Contact, droplet	Moderate	Use separate sinks for handwashing and disposal of contaminated fluids.	489, 629, 657 - 661
Showers	<i>Legionella</i>	Aerosol inhalation	Low	Provide sponge baths for HSCT patients; Avoid shower use in immunocompromised patient-care areas when <i>Legionella</i> is detected in facility water.	632
Dental unit water lines	<i>Pseudomonas</i> , <i>Legionella</i> , <i>Sphingomonas</i> , <i>Acinetobacter</i>	Contact	Low	Clean water systems according to system manufacturer's instructions.	613, 662 - 664
Ice baths for thermolulion catheters	<i>Ewingella</i> , <i>Staphylococcus</i>	Contact	Low	Use sterile water.	665, 666

(Continued next page)

Table 20 (continued). Water and Point-of-Use Fixtures

Reservoir	Associated Pathogen(s)	Transmission	Strength of Evidence ^b	Prevention and Control	References
Decorative fountains	<i>Legionella</i>	Aerosol inhalation	Low	Perform regular maintenance, water disinfection; avoid use in or near high-risk patient-care areas.	640
Eyewash stations	<i>Pseudomonas</i> , <i>Amoebae</i> <i>Legionella</i>	Contact	Low Minimum	Flush eyewash stations weekly; have sterile water available for eye flushes.	498, 667, 668
Toilets	Gram-negative bacteria	-	Minimum	Clean regularly; use good hand hygiene.	638
Flowers	Gram-negative bacteria, <i>Aspergillus</i>	-	Minimum	Avoid in ICUs and in immunocompromised patient-care settings.	495, 669, 670

a. Modified from reference 630. Used with permission of the publisher.

b. **Moderate:** occasional well-described outbreaks. **Low:** few well-described outbreaks. **Minimum:** actual infections not demonstrated.

b. Water Temperature and Pressure

Hot water temperature is usually measured at the point of use or at the point at which the water line enters equipment requiring hot water for proper operation.¹²⁰ Generally the hot water temperature in patient-care areas is no greater than 43°C (110°F),¹²⁰ and many states have adopted this temperature setting into their healthcare regulations and building codes. ASHRAE, however, has recommended higher settings.⁶³⁷ Steam jets or booster heaters are usually needed to meet the hot water temperature requirements in service areas of the hospital such as the kitchen (49°C [120°F]) or the laundry (71°C [160°F]).¹²⁰ Additionally, there may be other needs for water lines running a particular temperature specified by manufacturers of specific hospital equipment. Hot-water distribution systems serving patient-care areas are generally operated under constant recirculation to provide continuous hot water at each hot water outlet.¹²⁰ If a facility is or has a hemodialysis unit, then continuously circulated, cold treated water is provided to that unit.¹²⁰

To minimize the growth and persistence of gram-negative waterborne bacteria (e.g., thermophilic NTM, *Legionella* spp.),^{601, 671 - 677} cold water in healthcare facilities should be stored and distributed at temperatures below 20°C (68°F); hot water should be stored above 60°C (140°F) and circulated with a minimum return temperature of 51°C (124°F),⁶³⁷ or the highest temperature specified in state regulations and building codes. If the temperature setting of 51°C (124°F) is permitted, then installation of preset thermostatic mixing valves can help to prevent scalding. New shower systems in large buildings, hospitals, and nursing homes should be designed to permit mixing of hot and cold water near the shower head. The warm water section of pipe between the control valve and shower head should be self-draining. Where buildings cannot be retrofitted, other approaches to minimize the growth of *Legionella* spp. include periodically increasing the temperature to at least 66°C (150°F) at the point of use (i.e., faucets) or chlorinating followed by flushing the water.^{637, 678, 679} Systems should be inspected annually to ensure that thermostats are functioning properly.

Adequate water pressure ensures sufficient water supplies for: 1) direct patient care; 2) operation of water-cooled instruments and equipment [e.g., lasers, computer systems, telecommunications systems, automated endoscope reprocessors⁶⁸⁰]; 3) proper function of vacuum suctioning systems; 4) indoor climate control; and 5) fire protection systems. Maintaining adequate pressure also helps to insure the integrity of the piping system.

c. Infection Control Impact of Water System Maintenance and Repair

Corrective measures for water system failures have not been studied in well-designed experiments, but rather are based on empiric engineering and infection control principles.

Healthcare facilities can occasionally sustain intentional cut-offs by the municipal water authority to permit new construction project tie-ins and unintentional breaks in service when a water main breaks due to aging infrastructure or a construction accident. Vacuum breakers or other similar devices can prevent backflow of water in the facility's

distribution system during water-disruption emergencies.¹¹ To be prepared for such an emergency, all healthcare facilities need contingency plans that identify: 1) the total demand for potable water; 2) the quantity of replacement water (e.g., bottled water) required for a minimum of 24 hours when the water system is down; 3) mechanisms for emergency water distribution; and 4) procedures for correcting drops in water pressure that affect operation of essential devices and equipment that are driven or cooled by a water system.

Detailed current plans for hot and cold water piping systems should be readily available for maintenance and repair purposes in case of system problems. Opening potable water systems for repair or construction and subjecting systems to water-pressure changes can result in water discoloration and dramatic increases in the concentrations of *Legionella* spp. and other gram-negative bacteria. The maintenance of a chlorine residual at all points within the piping system also offers some protection from entry of contamination to the pipes in the event of an inadvertent cross-connection between potable and non-potable water lines. As a minimum preventive measure, ASHRAE recommends a thorough flushing of the system.⁶³⁷ High-temperature flushing or chlorination may also be appropriate strategies to decrease potentially high concentrations of waterborne organisms. The decision to pursue either of these remediation strategies, however, should be made on a case-by-case basis. If only a portion of the system is involved, high temperature flushing or chlorination may be used on only that portion of the system.⁶³⁷

When shock decontamination of hot water systems is necessary (e.g., after disruption due to construction, cross-connections), the hot water temperature should be raised to 71°C - 77°C (160°F - 170°F) and maintained at that level while progressively flushing each outlet around the system. A minimum flush time of 5 minutes has been recommended;³ the optimal flush time is not known, however, and longer flush times may be necessary.⁶⁸¹ The number of outlets that can be flushed simultaneously depends on the capacity of the water heater and the flow capability of the system. Appropriate safety procedures to prevent scalding are essential. When possible, flushing should be performed when the fewest building occupants are present (e.g., nights and weekends).

When thermal shock treatment is not possible, shock chlorination may provide an alternative.⁶³⁷ Experience with this method of decontamination is limited, however, and high levels of free chlorine can corrode metals. Chlorine should be added, preferably overnight, to achieve a free chlorine residual of at least 2 mg/L (2 ppm) throughout the system.⁶³⁷ This may require chlorination of the water heater or tank to levels of 20 - 50 mg/L (20 - 50 ppm). The pH of the water should be maintained between 7.0 and 8.0.⁶³⁷ After completion of the decontamination, recolonization of the hot water system is likely to occur unless proper temperatures are maintained or a procedure such as continuous supplemental chlorination is continued.

Interruptions of the water supply and sewage spills are situations which require immediate recovery and remediation measures to assure the health and safety of patients and staff.⁶⁸² When delivery of potable water through the municipal distribution system has been disrupted, the public water supplier must issue a “boil water” advisory if microbial contamination presents an immediate public health risk to customers. The hospital engineer should oversee the restoration of the water system in the facility and clear it for use when appropriate to do so. Hospitals must maintain a high level of surveillance for waterborne disease among patients and staff after the advisory is lifted.⁶¹⁹

Flooding from either external (e.g., from a hurricane) or internal sources (e.g., a water system break) usually results in property damage and a temporary loss of water and sanitation.^{683 - 685}

The JCAHO requires all hospitals have plans which address facility response for recovery from both internal and external disasters.⁶⁸⁶ The plans are required to address: 1) general emergency preparedness; 2) staffing; 3) regional planning among area hospitals; 4) emergency supply of potable water; 5) infection control and medical services needs; 6) climate control; and 7) remediation. The basic principles of structural recovery from flooding are similar to those for recovery from sewage contamination. Tables 21 - 23 summarize actions that will help to restore facility function and operations after water disruptions, sewage spills, and flooding. Medical records should be allowed to dry, and either photocopied or placed in plastic covers before returning them to the record. Moisture meters can be used to assess water-damaged structural materials.

Table 21. Recovery and Remediation Measures for Water-Related Emergencies^{683 - 695}

Potable Water Disruptions

Contingency plan items:

- Ensure access to plumbing network so that repairs can be made.
- Provide sufficient potable water, either from bottled sources or truck delivery.
- Post advisory notices against consuming tap water, ice, or beverages made with water.
- Rinse raw foods as needed in disinfected water.

Water treatment:

- Heat water to a rolling boil for 1 minute.

Remediation of the water system after the “boil water” advisory is rescinded:

- Flush fixtures (e.g., faucets, drinking fountains) and equipment for several minutes and restart.
 - Run water softeners through a regeneration cycle.
 - Drain, disinfect, and refill water storage tanks if needed.
 - Change pre-treatment filters and disinfect the dialysis water system.
-

Sewage Spills / Malfunction

Overall strategy:

- Move patients and clean/sterile supplies out of the area.
- Redirect traffic away from the area.
- Close the doors or use plastic sheeting to isolate the area prior to clean-up.
- Restore sewage system function first, then the potable water system (if both are malfunctioning).
- Remove sewage solids, drain the area, let dry, then clean with a detergent/disinfectant.

Remediation of the structure:

- Hard surfaces - drain and dry the area, then clean with a detergent/disinfectant.
- Carpeting, loose tiles, buckled flooring - remove and allow the support surface to dry; replace the items; wet down carpeting with a low-level disinfectant prior to removal.
- Wallboard and other porous structural materials - remove and replace if they cannot be cleaned and dried within 72 hours.

Furniture:

- Hard surface furniture (i.e., metal, plastic) - clean and allow to dry.
- Wood furniture - let dry, sand the wood surface, and reapply varnish.
- Cloth furniture - replace.

Electrical equipment:

- Replace if the item cannot be easily dismantled, cleaned, and reassembled.
-

An exception to these recommendations is made for hemodialysis units where water is further treated either by portable water treatment or large-scale water treatment systems usually involving reverse osmosis (RO). In the United States, greater than 98% of dialysis facilities use RO treatment for their water.⁶⁸⁷ It may be prudent, however, to change out pre-treatment filters and disinfect the system to prevent colonization of the RO membrane and microbial contamination down-stream of the pre-treatment filter.

Table 22. Contingency Planning - Flooding^{683 - 685}

General emergency preparedness:

- Ensure that emergency electrical generators are not located in flood-prone areas of the facility.
- Develop alternative strategies for moving patients, water containers, medical records, equipment, supplies, etc. if the elevators are inoperable.
- Establish in advance a centralized base of operations with batteries, flashlights, cellular phones.
- Ensure sufficient supplies of sandbags to sandbag the entrances and the area around boilers, incinerators, and generators.
- Establish alternative strategies for bringing core employees to the facility if high water prevents travel.

Staffing patterns:

- Temporarily reassign licensed staff as needed to critical care areas to provide manual ventilation and to perform vital assessments on patients.
- Designate a core group of employees to remain on site to keep all services operational if the facility remains open.
- Train all employees in emergency preparedness procedures.

Regional planning among area facilities for disaster management:

- Incorporate community support and involvement (e.g., media alerts, news, transportation).
- Develop in advance strategies for transferring patients as needed.
- Develop strategies for sharing supplies and providing essential services among participating facilities (e.g., central sterile department services, laundry).
- Identify sources for emergency provisions (e.g., blood, emergency vehicles, bottled water).

Medical services and infection control:

- Use waterless hand sanitizers in general patient-care areas.
- Postpone elective surgeries until full services are restored, or transfer these patients to other facilities.
- Consider using portable dialysis machines (water demand is less).
- Provide an adequate supply of tetanus and hepatitis A immunizations for patients and staff.

Climate control:

- Provide adequate water for cooling towers (may need to truck in, especially if the tower uses potable water).

Table 23. Water Demand in Healthcare Facilities During Water Disruption Emergencies

Potable Water

Drinking water
 Handwashing
 Cafeteria services
 Ice
 Manual flushing of toilets
 Patient baths, hygiene
 Hemodialysis
 Hydrotherapy
 Fire prevention (e.g., sprinkler systems)
 Surgery and critical care areas
 Laboratory services
 Laundry and central sterile departments (if these services cannot be arranged for elsewhere)
 Cooling towers^a

Bottled, Sterile Water

Surgical scrub
 Emergency surgical procedures
 Pharmaceutical preparations
 Patient-care equipment (e.g., ventilators, nebulizers) if electrical power is available

a. Some cooling towers may use a potable water source, but most units use non-potable water

4. Strategies for Controlling Waterborne Microbial Contamination

a. Supplemental Treatment of Water with Heat and/or Chemicals

In addition to using supplemental treatment methods as remediation measures after inadvertent contamination of water systems, healthcare facilities sometimes use special measures to control waterborne microorganisms on a sustained basis. This decision is most often associated with outbreaks of legionellosis and subsequent efforts to control legionellae,⁶⁸⁸ although some facilities have tried supplemental measures to better control thermophilic NTM.⁶⁰¹

The primary disinfectant for both cold and hot water systems is chlorine. However, chlorine residuals are expected to be low, and possibly nonexistent, in hot water tanks due to extended retention time in the tank and elevated water temperature. Flushing, especially that which removes sludge from the bottom of the tank, probably provides the most effective treatment of water systems. Unlike the situation for disinfecting cooling towers, there are no equivalent recommendations for potable water systems, although specific intervention strategies have been published.^{389, 689} The principal approaches to disinfection of potable systems are heat flushing using temperatures 71°C - 77°C (160°F - 170°F), hyperchlorination, and physical cleaning of hot water tanks.^{3, 389, 637} Potable systems are easily recolonized and may require continuous intervention such as raising of hot water temperatures or continuous chlorination.^{389, 679}

Some hospitals with hot water systems identified as the source of *Legionella* spp. have decontaminated their systems by pulse (one-time) thermal disinfection/superheating and hyperchlorination.^{679, 681, 690, 691} After either of these procedures, hospitals either maintain their hot water at $\geq 51^{\circ}\text{C}$ ($\geq 124^{\circ}\text{F}$) or $< 20^{\circ}\text{C}$ ($< 68^{\circ}\text{F}$) at the tap or chlorinate their hot water to achieve 1-2 mg/L (1-2 ppm) of free residual chlorine at the tap.^{26, 436, 677 - 679, 692, 693} Additional measures (e.g., physical cleaning or replacement of hot-water storage tanks, water heaters, faucets, and showerheads) may be required to help eliminate accumulations of scale and sediment that protect organisms from the biocidal effects of heat and chlorine.^{398, 679} Alternative methods for controlling and eradicating legionellae in water systems (e.g., treating water with ozone, UV light, heavy metal ions [i.e., copper/silver ions]) have limited the growth of legionellae under laboratory and/or operating conditions.^{694 - 705} However, results from a recent study suggest that legionellae develop tolerance to copper/silver ion treatment during extended application (≥ 4 years);⁷⁰⁶ further studies on the long-term efficacy of these treatments are needed, however, before they can be considered standard precautions.

Renewed interest in the use of chloramines stems from concerns about adverse health effects associated with disinfectants and disinfection by-products.⁷⁰⁷ Monochloramine usage minimizes the formation of disinfection by-products, including trihalomethanes and haloacetic acids. Monochloramines can also reach distal points in a water system and can penetrate into bacterial biofilms more effectively than free chlorine.⁷⁰⁸ It should be noted that monochloramine use is limited to municipal water treatment plants and is currently not available to healthcare facilities as a supplemental water treatment approach. A recent study indicated that 90% of Legionnaires' disease outbreaks associated with drinking water could have been prevented if monochloramine rather than free chlorine was used for residual disinfection.⁷⁰⁹ In a retrospective comparison of healthcare-associated Legionnaires' disease incidence in Central Texas hospitals, the same research group documented an absence of cases in facilities located in communities with monochloramine-treated municipal water.⁷¹⁰

Additional filtration of potable water systems is not routinely necessary. Filters are used in water lines in dialysis units, however, and may be inserted into the lines for specific equipment (e.g., endoscope washer/disinfectors) for the purpose of providing bacteria-free water for instrument reprocessing. Additionally, a reverse osmosis (RO) unit is usually added to the distribution system leading to PE areas.

b. Primary Prevention of Legionnaires' Disease (No Cases Identified)

The primary and secondary environmental infection control strategies described below pertain to healthcare facilities without transplant. Infection control measures specific to PE or transplant units (patient-care areas housing patients at the highest risk for morbidity and mortality due to *Legionella* spp. infection) are described in the subsection entitled "Preventing Legionnaires' Disease in Protective Environments."

Healthcare facilities use at least two general strategies to prevent healthcare-associated legionellosis when no cases or only sporadic cases have been detected. The first is an environmental surveillance approach, with periodic culturing of water samples from the hospital's potable water system to monitor for *Legionella* spp.^{711 - 714} If any sample is culture-

positive, diagnostic testing is recommended for all patients with healthcare-associated pneumonia.^{712, 713} In-house testing is recommended for facilities with transplant programs. If $\geq 30\%$ of the samples are culture-positive for *Legionella* spp., decontamination of the facility's potable water system is recommended.⁷¹² The premise for this approach is that no cases of healthcare-associated legionellosis can occur if *Legionella* spp. are not present in the potable water system, and, conversely, cases of healthcare-associated legionellosis could potentially occur if *Legionella* spp. are cultured from the water.^{26, 715} Physicians informed that the hospital's potable water system is culture-positive for *Legionella* spp. are more likely to order diagnostic tests for legionellosis.

A potential advantage of the environmental surveillance approach is that periodic culturing of water is less costly than routine laboratory diagnostic testing for all patients who have healthcare-associated pneumonia. The main argument against this approach is that, in the absence of cases, the relationship between water culture results and legionellosis risk remains undefined.³ *Legionella* spp. can be present in the water systems of buildings,⁷¹⁶ often without being associated with known cases of disease.^{436, 675, 717} In a study of 84 hospitals in Québec, 68% of the water systems were found to be colonized with *Legionella* spp., and 26% were colonized at greater than 30% of sites sampled; cases of Legionnaires' disease, however, were infrequently reported from these hospitals.⁶⁷⁵

Other factors also argue against environmental surveillance. Interpretation of results from periodic water culturing might be confounded by differing results among the sites sampled in a single water system and by fluctuations in the concentration of *Legionella* spp. at the same site.^{677, 718} In addition, the risk for illness after exposure to a given source might be influenced by a number of factors other than the presence or concentration of organisms; these factors include: 1) the degree to which contaminated water is aerosolized into respirable droplets; 2) the proximity of the infectious aerosol to the potential host; 3) the susceptibility of the host; and 4) the virulence properties of the contaminating strain.^{719 - 721} Thus, data are insufficient to assign a level of disease risk even on the basis of the number of colony-forming units detected in samples from areas for immunocompetent patients. Conducting environmental surveillance would obligate hospital administrators to initiate water-decontamination programs if *Legionella* spp. are identified. Because of these problems, periodic monitoring of water from the hospital's potable water system and from aerosol-producing devices is not widely recommended in facilities that have not experienced cases of healthcare-associated legionellosis.^{637, 722}

The second strategy to prevent and control healthcare-associated legionellosis is a clinical approach in which providers maintain a high index of suspicion for legionellosis and order appropriate diagnostic tests (i.e., culture, urine antigen, direct fluorescent antibody [DFA] serology) for patients with healthcare-associated pneumonia who are at high risk for legionellosis and its complications.^{436, 723, 724} Testing autopsy specimens can be included in this strategy should a death due to healthcare-associated pneumonia occur. Identification of one case of definite or two cases of possible healthcare-associated Legionnaires' disease prompts an epidemiologic investigation for a hospital source of *Legionella* spp. This may involve culturing the facility's water for *Legionella* spp. Routine maintenance of cooling towers and using only sterile water for the filling and terminal rinsing of nebulization devices and ventilation equipment help to minimize potential sources of contamination.

c. Secondary Prevention of Legionnaires' Disease (With Identified Cases)

The indications for a full-scale environmental investigation to search for and subsequently decontaminate identified sources of *Legionella* spp. in healthcare facilities without transplant units have not been clarified, and these indications probably differ depending on the facility. Case categories for healthcare-associated Legionnaires' disease in facilities without transplant units include definite cases (i.e., laboratory-confirmed cases of legionellosis that occur in patients who have been hospitalized continuously for ≥ 10 days before the onset of illness) and possible cases (i.e., laboratory-confirmed infections that occur 2 - 9 days after hospital admission).³ In settings in which as few as 1 - 3 healthcare-associated cases are recognized over several months, intensified surveillance for Legionnaires' disease has frequently identified numerous additional cases.^{385, 391, 394, 430, 705, 723} This finding suggests the need for a low threshold for initiating an investigation after laboratory confirmation of cases of healthcare-associated legionellosis. When developing a strategy for responding to such an identification, however, infection control personnel should consider the level of risk for healthcare-associated acquisition of, and mortality from, *Legionella* spp. infection at their particular facility.

An epidemiologic investigation conducted to determine the source of *Legionella* spp. involves several important steps

(Table 24). Laboratory assistance is important in supporting epidemiologic evidence of a link between human illness and a specific environmental source.⁷²⁵ Strain determination from subtype analysis is most frequently used in these investigations.^{396, 726- 728} Once the environmental source is established and confirmed with laboratory support, supplemental water treatment strategies can be initiated as appropriate.

Table 24. Steps in an Epidemiologic Investigation for Legionellosis

-
- Review medical and microbiologic records.
 - Initiate active surveillance to identify all recent or ongoing cases.
 - Develop a line listing of cases by time, place, and person.
 - Determine the type of epidemiologic investigation needed for assessing risk factors.
 - Case-control study
 - Cohort study
 - Gather and analyze epidemiologic information.
 - Risk factors among potential environmental exposures (e.g., showers, cooling towers, respiratory-therapy equipment, etc.)
 - Collect water samples.
 - From environmental sources implicated by epidemiologic investigation
 - Other aerosolized water sources
 - Subtype strains of *Legionella* spp. cultured from both patients and environmental sources.
 - Review autopsy records and include autopsy specimens in diagnostic testing.
-

The decision to search for hospital environmental sources of *Legionella* spp. and the choice of procedures to eradicate such contamination are based on several considerations: 1) the hospital's patient population; 2) the cost of an environmental investigation and institution of control measures to eradicate *Legionella* spp. from the water supply,^{729, 730} and 3) the differential risk, based on host factors, for acquiring healthcare-associated legionellosis and developing severe and fatal infection.

d. Preventing Legionnaires' Disease in Protective Environments

This subsection outlines infection control measures applicable to those healthcare facilities providing care to severely neutropenic patients, as indigenous microorganisms in the tap water of these facilities may pose problems for such patients. These measures, summarized in Table 25, are designed to prevent the generation of potentially infectious aerosols from water and the subsequent exposure of PE patients or other immunocompromised patients (e.g., transplant patients). Infection control measures that address the use of water with medical equipment such as ventilators, nebulizers, and equipment humidifiers are described in other guidelines.³

When one case of laboratory-confirmed, healthcare-associated Legionnaires' disease is identified in a patient in PE (i.e., an inpatient in PE for all or part of the 2 - 10 days prior to onset of illness), or if two or more cases of laboratory-confirmed cases occur among patients who had visited an outpatient PE setting, the hospital should report the cases to the local and state health departments and initiate a thorough epidemiologic and environmental investigation to determine the likely environmental sources of *Legionella* spp.⁹ The source of *Legionella* should be decontaminated or removed. Isolated cases may be difficult to investigate. Because transplant recipients are at much higher risk for disease and death from legionellosis compared to most other hospitalized patients, periodic culturing for *Legionella* spp. in water samples from the PE unit's potable water supply may be considered as part of an overall strategy to prevent Legionnaires' disease in PE units.^{9, 430} The optimal methodology (i.e., frequency, number of sites) for environmental surveillance cultures in PE units has not been determined, and the cost-effectiveness of this strategy has not been evaluated. Because transplant recipients are at high risk of Legionnaires' disease and there are no data to determine a safe concentration of legionellae organisms in potable water, the goal, if environmental surveillance for *Legionella* spp. is undertaken, should be to maintain water systems with no detectable organisms.^{9, 430} Culturing for legionellae may be used to assess the effectiveness of water treatment or decontamination methods, which provides a benefit to both patients and healthcare workers.⁷³¹

Table 25. Additional Measures to Prevent Exposure of High-Risk Patients to Waterborne Pathogens

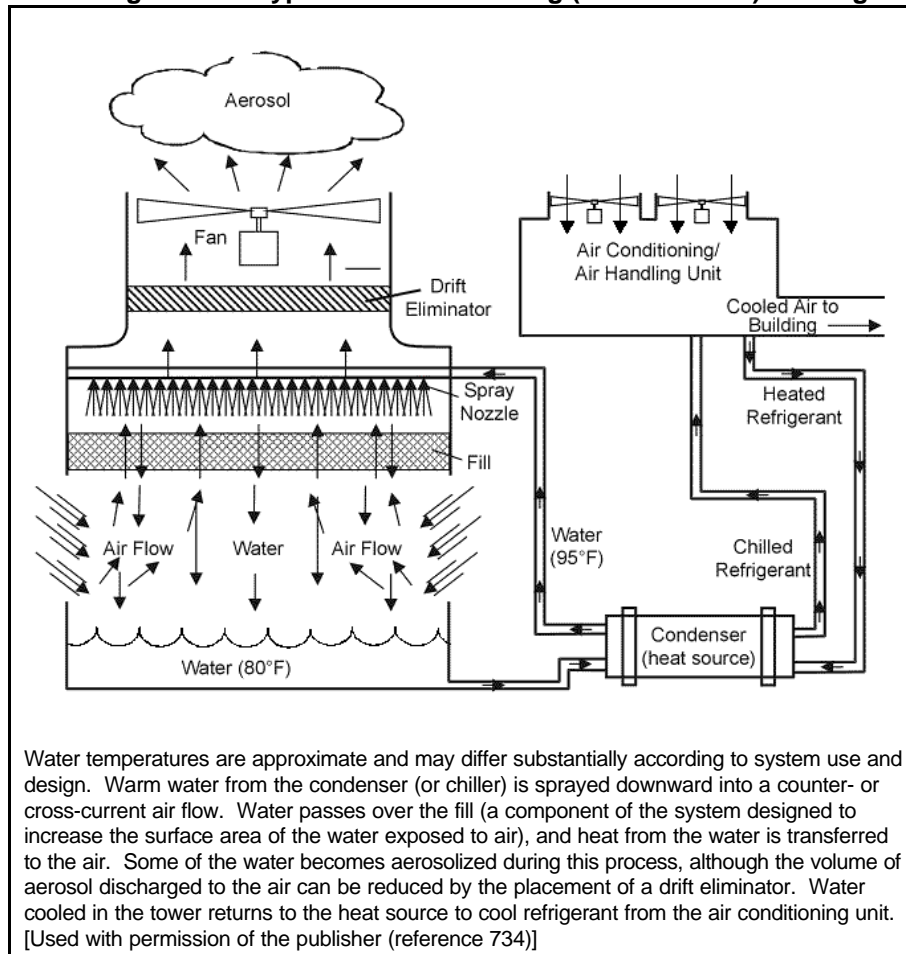
	Reference(s)
1. Restrict patients from taking showers if water is contaminated with <i>Legionella</i> spp.	393, 411, 630, 631, 634
2. Use water which is not contaminated with <i>Legionella</i> spp. for patient sponge baths.	9
3. Provide sterile water for drinking, tooth brushing, or for flushing nasogastric tubes.	9, 411
4. Perform supplemental treatment of the water for the unit.	698
5. Consider periodic monitoring (culturing) of the unit water supply for <i>Legionella</i> spp.	9, 430
6. Remove shower heads and faucet aerators monthly for cleaning.	637
7. Use a 1:100 solution of chlorine bleach to disinfect shower heads and aerators.	637
8. Do not use large-volume room air humidifiers that create aerosols unless these are subjected to high-level disinfection daily and filled with sterile water.	3
9. Eliminate water-containing bath toys (associated with <i>Pseudomonas</i>).	30

Protecting patient-care devices and instruments from inadvertent tap water contamination during room cleaning procedures is also important in any immunocompromised patient care area. In a recent outbreak of gram-negative bacteremias among open-heart-surgery patients, pressure-monitoring equipment which was assembled and left uncovered overnight prior to the next day's surgeries was inadvertently contaminated with mists and splashing water from a hose-disinfectant system used for cleaning.⁷³²

5. Cooling Towers and Evaporative Condensers

Modern healthcare facilities maintain indoor climate control during the summer by use of cooling towers for large facilities or evaporative condensers for smaller buildings. A cooling tower is a wet-type, evaporative heat transfer device used to discharge to the atmosphere waste heat from a building's air conditioning condensers (Figure 5).^{733, 734} Warm water from air-conditioning condensers is piped to the cooling tower where it is sprayed downward into a counter- or cross-current air flow. To accelerate heat transfer to the air, the water passes over the fill, which either breaks water into droplets or causes it to spread into a thin film.^{733, 734} Most systems use fans to move air through the tower, although some large industrial cooling towers rely on natural draft circulation of air. The cooled water from the tower is piped back to the condenser where it again picks up heat generated during the process of chilling the system's refrigerant. The water is cycled back to the cooling tower to be cooled. Closed-circuit cooling towers and evaporative condensers are also evaporative heat transfer devices. In these systems, the process fluid (i.e., a liquid such as water, ethylene glycol/water mixture, oil, etc. or a condensing refrigerant) does not directly contact the cooling air, but is contained inside a coil assembly.⁶³⁷

Figure 5. Diagram of a Typical Air Conditioning (Induced Draft) Cooling Tower.^a



Cooling towers and evaporative condensers incorporate inertial stripping devices called drift eliminators to remove water droplets generated within the unit. While the effectiveness of these eliminators varies significantly based on their design and condition, some water droplets in the size range of $<5 \mu\text{m}$ will likely leave the unit, and some larger droplets leaving the unit may be reduced to $\leq 5 \mu\text{m}$ by evaporation. Thus, even with proper operation, a cooling tower or evaporative condenser can generate and expel respirable water aerosols. If either the water in the unit's basin or the make-up water (added to replace water lost to evaporation) contains *Legionella* spp. or other waterborne microorganisms, these organisms can be aerosolized and dispersed from the unit.⁷³⁵ Clusters of both Legionnaires' disease and Pontiac fever have been traced to exposure to infectious water aerosols originating from cooling towers and evaporative condensers contaminated with *Legionella* spp. Although the majority of these outbreaks have been community-acquired episodes of pneumonia,^{736 - 743} there have been instances of healthcare-associated Legionnaires' disease linked to cooling tower aerosol exposure.^{390, 391} Contaminated aerosols from cooling towers on hospital premises gained entry to the buildings either through open windows or via air handling system intakes located near the tower equipment.

Cooling towers and evaporative condensers provide ideal ecological niches for *Legionella* spp. The typical temperature of the water in cooling towers ranges from $29^{\circ}\text{C} - 35^{\circ}\text{C}$ ($85^{\circ}\text{F} - 95^{\circ}\text{F}$), although temperatures can be above 49°C (120°F) and below 21°C (70°F) depending on system heat load, ambient temperature and operating strategy.⁶³⁷ An Australian study of cooling towers found that legionellae colonized or multiplied in towers with basin temperatures above 16°C (60.8°F), and multiplication became explosive at temperatures above 23°C (73.4°F).⁷⁴⁴ Water temperature in closed circuit cooling towers and evaporative condensers is similar to that in cooling towers. Considerable variation in the piping arrangement occurs. Stagnant areas or dead legs may be difficult to clean or penetrate with biocides.

Several documents address the routine maintenance of cooling towers, evaporative condensers, and whirlpool spas.^{637, 745}

- 748 They suggest following manufacturer's recommendations for cleaning and biocide treatment of these devices; all healthcare facilities should provide proper maintenance for their cooling towers and evaporative condensers, even in the absence of *Legionella* spp. A general protocol for cleaning cooling towers is given in Appendix C. Since cooling towers and evaporative condensers may be shut down during periods when air conditioning is not needed, it is important to perform this maintenance cleaning and treatment before starting up the system for the first time in the season.⁷⁴³ Emergency decontamination protocols describing cleaning procedures and hyperchlorination for cooling towers have been developed for towers implicated in the transmission of legionellosis.^{747, 748}

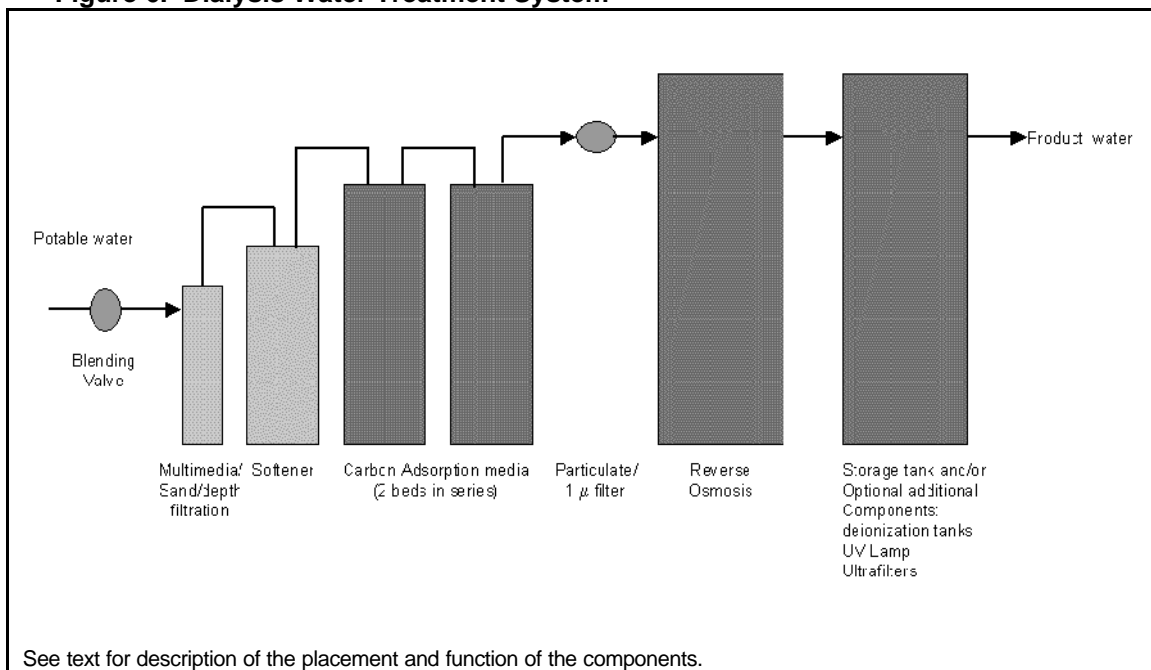
6. Dialysis Water Quality and Dialysate

a. Rationale for Water Treatment in Hemodialysis

Hemodialysis, hemofiltration, and hemodiafiltration require special water treatment processes to prevent adverse patient outcomes of dialysis therapy due to improper formulation of dialysate with water containing high levels of certain chemical or biological contaminants. The Association for the Advancement of Medical Instrumentation (AAMI) has established chemical and microbiologic standards for the water used to prepare dialysate, substitution fluid, or to reprocess hemodialyzers for renal replacement therapy.^{749 - 752} The AAMI standards address: 1) equipment and processes used to purify water for the preparation of concentrates and dialysate, and the reprocessing of dialyzers for multiple use; and 2) the devices used to store and distribute this water. Future revisions to these standards may include hemofiltration and hemodiafiltration.

Water treatment systems used in hemodialysis use several physical and/or chemical processes either singly or in combination. A schematic diagram of basic water treatment components in dialysis is given in Figure 6. These systems may be portable units or large systems which feed several rooms. In the United States, more than 97% of maintenance hemodialysis facilities use reverse osmosis (RO) alone or in combination with deionization.⁷⁵³ Many acute-care facilities use portable hemodialysis machines with attached portable water treatment systems that use either deionization or RO. These machines were exempted from earlier versions of AAMI recommendations, but given current knowledge about toxic exposures to and inflammatory processes in patients new to dialysis, these should now come into compliance with current AAMI recommendations for hemodialysis water and dialysate quality.^{749, 750} Previous recommendations had been based on the assumption that acute-care patients did not experience the same degree of adverse effects from short-term, cumulative exposures to either chemicals or microbiologic agents present in hemodialysis fluids, compared to the risks encountered by patients during chronic, maintenance dialysis.^{749, 750} Additionally, the JCAHO is now reviewing inpatient dialysis (acute and maintenance) for compliance with the AAMI standards and recommended practices.

Figure 6. Dialysis Water Treatment System



Neither the water used to prepare dialysate nor the dialysate itself needs to be sterile, but tap water cannot be used without additional treatment. Infections due to rapid-growing NTM (e.g., *Mycobacterium chelonae*, *M. abscessus*) present a potential risk to hemodialysis patients, especially those in hemodialyzer reuse programs, if disinfection procedures to inactivate mycobacteria in the water (low-level disinfection) and the hemodialyzers (high-level disinfection) are inadequate.^{31, 32, 610} Other factors relating to microbial contamination in dialysis systems could involve the water treatment system, the water and dialysate distribution systems, and in some cases, the type of hemodialyzer.^{642, 643, 754 - 759} Understanding the various factors and their influence on contamination levels is the key to preventing high levels of microbial contamination in dialysis therapy.

In several studies, pyrogenic reactions were shown to be caused by lipopolysaccharide or endotoxin associated with gram-negative bacteria.^{754, 760 - 763} Early studies demonstrated that parenteral exposure to endotoxin at a concentration of 1 ng/kg body weight/hour was the threshold dose for producing pyrogenic reactions in humans, and that the relative potencies of endotoxin differ by bacterial species.^{764, 765} Gram-negative water bacteria (e.g., *Pseudomonas* spp.) have been shown to multiply rapidly in a variety of hospital-associated fluids that can be used as supply water for hemodialysis (e.g., distilled water, deionized water, RO water, softened water) and in dialysate (a balanced salt solution made with this water).⁷⁶⁶ Several studies have demonstrated that the attack rates of pyrogenic reactions are related directly to the number of bacteria in dialysate.^{642, 643, 767} These studies provided the rationale for setting the heterotrophic bacteria standards in the first AAMI hemodialysis guideline at #2,000 colony forming units per milliliter (CFU/mL) in dialysate and one log lower (#200 CFU/mL) for the water used to prepare dialysate.^{644, 749} If the level of bacterial contamination exceeded 200 CFU/mL in water, this level could be amplified in the system and effectively constitute a high inoculum for dialysate at the start of a dialysis treatment.^{767, 768} Pyrogenic reactions did not appear to occur when the level of contamination was below 2,000 CFU/mL in dialysate unless the source of the endotoxin was exogenous to the dialysis system (i.e., present in the community water supply). Endotoxins in a community water supply have been linked to the development of pyrogenic reactions among dialysis patients.⁷⁵⁴

The issue as to whether endotoxin actually crosses the dialyzer membrane is controversial. Several investigators have shown that bacteria, growing in dialysate, generated products that could cross the dialysis membrane.^{769, 770} Gram-negative bacteria growing in dialysate have been shown to produce endotoxins that in turn stimulated the production of anti-endotoxin antibodies in hemodialysis patients.^{761, 771} These data suggest that bacterial endotoxins, although relatively large molecules, do indeed cross dialysis membranes, either intact or as fragments. The use of the very permeable membranes known as high-flux membranes (which allow large molecules [e.g., β_2 microglobulin] to traverse the membrane) increases the potential for passage of endotoxins into the blood path. Several studies support this contention. In one such study, an increase in plasma endotoxin concentrations during dialysis was observed when patients were dialyzed against dialysate containing 10^3 - 10^4 CFU/mL *Pseudomonas* spp.⁷⁷² *In vitro* studies using both radiolabeled lipopolysaccharide and biological assays have demonstrated that biologically active substances derived from bacteria found in dialysate can cross a variety of dialysis membranes.^{762, 773 - 776} Patients treated with high-flux membranes are reported to have higher levels of anti-endotoxin antibodies than normal subjects or patients treated with conventional membranes.⁷⁷⁷ Finally, since 1989, 19% - 22% of dialysis centers have reported pyrogenic reactions in the absence of septicemia.^{778, 779}

Investigations of adverse outcomes among patients using reprocessed dialyzers demonstrated a greater risk of developing pyrogenic reactions when the water used to reprocess these devices contained > 6 ng/mL endotoxin and $> 10^4$ CFU/mL bacteria.⁷⁸⁰ In addition to the variability in endotoxin assays, there are also host factors involved in determining whether a patient will mount a response to endotoxin.⁷⁶³ Outbreak investigations of pyrogenic reactions and bacteremias associated with hemodialyzer reuse have demonstrated that pyrogenic reactions are prevented once the endotoxin level in the water used to reprocess the dialyzers is returned to below the AAMI standard level.⁷⁸¹

Reuse of dialyzers, use of bicarbonate dialysate, high-flux dialyzer membranes, or high-flux dialysis may increase the potential for pyrogenic reactions if the water in the dialysis setting does not meet standards.^{756 - 758} Although investigators have not been able to demonstrate endotoxin transfer across dialysis membranes,^{763, 782, 783} the preponderance of reports now supports the ability of endotoxin to transfer across at least some high-flux membranes under some operating conditions. In addition to the acute risk of pyrogenic reactions, there is increasing indirect evidence that chronic exposure to low amounts of endotoxin may play a role in some of the long-term complications of

hemodialysis therapy. Patients treated with ultrafiltered dialysate for 5-6 months have demonstrated a decrease in serum β 2-microglobulin concentrations and a decrease in markers of an inflammatory response.^{784 - 786} In studies of longer duration, use of microbiologically ultrapure dialysate has been associated with a decreased incidence of β 2-microglobulin-associated amyloidosis.^{787, 788}

The current AAMI standard does not provide for endotoxin testing of all dialysis fluids. Only water that is used for the reprocessing of hemodialyzers has an endotoxin limit of 5 endotoxin units per milliliter (EU/mL [Table 26]), and the current standard recommends this as a choice. CDC has advocated monthly endotoxin testing along with microbiological assays of water since endotoxin activity may not correspond to the total heterotrophic plate counts.⁷⁸⁹ Consequently, the proposed revision to the AAMI standard may impose an upper limit on the endotoxin content of all water for hemodialysis applications. A level of 2 EU/mL was chosen as the upper limit for endotoxin because this level is easily achieved with contemporary water treatment systems using RO and/or ultrafiltration. Because 48 hours can elapse between the time of sampling water for the determination of microbial contamination and the time when results are received, and because bacterial proliferation can be rapid, action levels for microbial counts and endotoxin concentrations are also being considered in this revision of the standard. These will allow users to initiate corrective action before levels exceed the maximum levels established by the standard.

Table 26. Microbiological Limits for Hemodialysis Fluids^{750, 752}

Hemodialysis Fluid	Maximum Total Heterotrophs (CFU/mL)	Maximum Endotoxin Level (EU/mL)
Water		
Used to Prepare Dialysate	200	No Standard
Used for Reprocessing	200	5
Dialysate	2,000	No Standard
Proposed AAMI Standard		
Water	200	2
Dialysate	Not Determined	Not Determined

In hemodialysis, the net movement of water is from the blood to the dialysate, although within the dialyzer there may be local movement of water from the dialysate to the blood through the phenomenon of back-filtration, particularly in dialyzers with highly permeable membranes.⁷⁹⁰ In contrast, hemofiltration and hemodiafiltration feature infusion of large volumes of electrolyte solution (20 - 70 L) into the blood. Increasingly, this electrolyte solution is being prepared on-line from water and concentrate. Because of the large volumes of fluid infused, AAMI considered the necessity of setting more stringent requirements for water to be used in this application, but has not yet established these due to lack of expert consensus. On-line hemofiltration and hemodiafiltration systems use sequential ultrafiltration as the final step in the preparation of infusion fluid. Several experts from AAMI felt that these point-of-use ultrafiltration systems should be capable of further reducing the bacteria and endotoxin burden of solutions prepared from water meeting the requirements of the AAMI standard to a safe level for infusion.

b. Microbial Control Strategies

The strategy for controlling massive accumulations of gram-negative water bacteria and NTM in dialysis systems primarily involves preventing their growth through proper disinfection of water treatment system and hemodialysis machines. Gram-negative water bacteria, their associated lipopolysaccharides (bacterial endotoxins), and NTM ultimately come from the community water supply, and levels of these bacteria can be amplified depending on the water treatment system, dialysate distribution system, type of dialysis machine, and method of disinfection (Table 27).^{610, 754, 791} Control strategies are designed to reduce levels of microbial contamination in water and dialysis fluid to relatively low levels but not to completely eradicate it.

Two components of hemodialysis water distribution systems – pipes and storage tanks – can serve as reservoirs of microbial contamination. Hemodialysis systems frequently use pipes that are wider and longer than are needed to handle the required flow. This slows the fluid velocity and increases both the total fluid volume and the wetted surface area of the system. Gram-negative bacteria in fluids remaining in pipes overnight multiply rapidly and colonize the wet surfaces, producing bacterial populations and endotoxin quantities in proportion to the volume and surface area. Such colonization results in formation of protective biofilm that is difficult to remove and protects the bacteria from disinfection.⁷⁹² Routine low-level disinfection of the pipes on a weekly basis can help to control bacterial contamination of the distribution system. Additional measures to protect pipes from contaminations include: 1) situating all outlet taps at equal elevation and at the highest point of the system so that the disinfectant cannot drain from pipes by gravity before adequate contact time has elapsed; and 2) eliminating rough joints, dead-end pipes, and unused branches and taps that can trap fluid and serve as reservoirs of bacteria capable of continuously inoculating the entire volume of the system.⁷⁶⁰

A storage tank in the distribution system greatly increases the volume of fluid and surface area available and can serve as a niche for water bacteria. Storage tanks are therefore not recommended for use in dialysis systems unless they are frequently drained and adequately disinfected, including scrubbing the sides of the tank to remove bacterial biofilm. An ultrafilter should be used distal to the storage tank.^{768, 793}

Microbiologic sampling of dialysis fluids is recommended because gram-negative bacteria can proliferate rapidly in water and dialysate in hemodialysis systems; high levels of these organisms place patients at risk of pyrogenic reactions or healthcare-associated infection.^{643, 644, 768}

Table 27. Factors Influencing Microbial Contamination in Hemodialysis Systems

Factors	Comments
<u>Water supply</u>	
Source of community water	
Groundwater	Contains endotoxin and bacteria
Surface water	Contains high levels of endotoxin and bacteria
<u>Water treatment at dialysis center</u>	
None	Not recommended
Filtration	
Prefilter	Particulate filter to protect equipment; does not remove microorganisms
Absolute filter (depth or membrane)	Removes bacteria but, unless changed frequently or disinfected, bacteria will accumulate and grow through filter; acts as significant reservoir of bacteria and endotoxin
Activated carbon filter	Removes organics and available chlorine or chloramine; significant reservoir of water bacteria and endotoxin
<u>Water treatment devices</u>	
Deionization/Ion-exchange softener	Both softeners and de-ionizers are significant reservoirs of bacteria and do not remove endotoxin
Reverse osmosis	Removes bacteria and endotoxin, but must be disinfected; operates at high water pressure
Ultraviolet light	Kills some bacteria, but there is no residual, and ultraviolet-resistant bacteria can develop if not properly maintained
Ultrafilter	Removes bacteria and endotoxin; operates on normal line pressure; can be positioned distal to de-ionizer; must be disinfected
<u>Water and dialysate distribution system</u>	
Distribution pipes	
Size	Oversized diameter and length decrease fluid flow and increase bacteria reservoir for both treated water and centrally prepared dialysate
Construction	Rough joints, dead ends, and unused branches can act as bacterial reservoirs
Elevation	Outlet taps should be located at highest elevation to prevent loss of disinfectant
Storage tanks	Undesirable because they act as reservoir of water bacteria; if present, must be routinely scrubbed and disinfected
<u>Dialysis machines</u>	
Single-pass	Disinfectant should have contact with <i>all</i> parts of machine that are exposed to water or dialysis fluid.
Recirculating single-pass, or recirculating (batch)	Recirculating pumps and machine design allow for massive contamination levels if not properly disinfected. Overnight chemical germicide treatment recommended.

Healthcare facilities are advised to sample dialysis fluids at least monthly using standard microbiological assay methods for waterborne microorganisms.^{750, 753, 759, 794 - 796} Water used to reprocess hemodialyzers for reuse on the same patient should also be tested for bacterial endotoxin on a monthly basis.^{789, 797} Information about water sampling methods for dialysis is provided in Appendix C.

Cross-contamination of dialysis machines and inadequate disinfection measures can facilitate the spread of waterborne organisms to patients. Steps should be taken to ensure that dialysis equipment is performing correctly and that all connectors, lines, and other components are specific for the equipment, in good repair, and properly in place. A recent outbreak of gram-negative bacteremias among dialysis patients was attributed to faulty valves in a drain port of the machine that allowed backflow of saline used to flush the dialyzer before patient use.^{798, 799} This backflow contaminated

the drain priming connectors, which in turn contaminated the blood lines and exposed the patients to high concentrations of gram-negative bacteria. Environmental infection control in dialysis settings also includes low-level disinfection of housekeeping surfaces and spot decontamination of spills of blood (see the Environmental Services portion of Part I of this guideline for further information).

c. Peritoneal Dialysis Issues

Peritoneal dialysis (PD), most commonly administered as continuous ambulatory peritoneal dialysis (CAPD) and continual cycling peritoneal dialysis (CCPD), is the third most common treatment for end-stage renal disease (ESRD) in the United States, accounting for 12% of all dialysis patients.⁸⁰⁰ Peritonitis is the most important complication of CAPD, with coagulase-negative staphylococci the most clinically significant causative organisms.⁸⁰¹ Other organisms that have been found to produce peritonitis include *Staphylococcus aureus*, *Mycobacterium fortuitum*, *M. mucogenicum*, *Stenotrophomonas maltophilia*, *Burkholderia cepacia*, *Corynebacterium jeikeium*, *Candida* spp., and other fungi.^{802 - 810} Substantial morbidity is associated with peritoneal dialysis infections. Removal of peritoneal dialysis catheters is usually required for treatment of peritonitis caused by fungi, NTM, or other bacteria that are not cleared within the first several days of effective antimicrobial treatment. Furthermore, recurrent episodes of peritonitis may lead to fibrosis and loss of the dialysis membrane.

Many reported episodes of peritonitis are associated with exit-site or tunneled catheter infections. Risk factors for the development of peritonitis in PD patients include: 1) under dialysis; 2) immune suppression; 3) prolonged antimicrobial treatment; 4) patient age [more infections in younger patients and older hospitalized patients]; 5) length of hospital stay; and 6) lower hypoalbuminemia.^{804, 811, 812} There has been some concern about infection risk related to the use of automated cyclers in both inpatient and outpatient settings. However, studies suggest that PD patients who use automated cyclers have much lower infection rates.⁸¹³ One study noted that a closed-drainage system reduced the incidence of system-related peritonitis among intermittent peritoneal dialysis (IPD) patients from 3.6 to 1.5 cases/100 patient days.⁸¹⁴ The association of peritonitis with management of spent dialysate fluids requires additional study. At present, it is prudent to ensure that the tip of the waste line is not submerged beneath the water level in a toilet or in a drain.

7. Ice Machines and Ice

Microorganisms may be present in ice, ice-storage chests and ice-making machines. The two main sources of microorganisms in ice are the potable water from which it is made and a transferral of organisms from hands (Table 28). Ice from contaminated ice machines has been associated with patient colonization, blood stream infections, pulmonary and gastrointestinal illnesses, and pseudoinfections.^{580, 581, 653, 654, 815, 816} Microorganisms in ice can secondarily contaminate clinical specimens and medical solutions which require cold temperatures for either transport or holding.^{579, 598} An outbreak of surgical site infections was interrupted when sterile ice was used in place of tap water ice to cool cardioplegia solutions.⁵⁷⁹

Table 28. Sources of Microorganisms in Ice and Ice Machines

<i>From Potable Water</i>	<i>References</i>
<i>Legionella</i> spp.	655, 653, 817, 818
Non-tuberculous mycobacteria (NTM)	580, 581, 819
<i>Pseudomonas aeruginosa</i>	819
<i>Burkholderia cepacia</i>	819, 820
<i>Stenotrophomonas maltophilia</i>	820
<i>Flavobacterium</i> spp.	820
<i>From Fecally-Contaminated Water</i>	
Norwalk virus	821, 822, 823
<i>Giardia lamblia</i>	824
<i>From Hand-Transfer of Organisms</i>	
<i>Acinetobacter</i> spp.	819
Coagulase-negative staphylococci	819
<i>Salmonella enteritidis</i>	825
<i>Cryptosporidium parvum</i>	654

In a study comparing the microbial populations of hospital ice machines with organisms recovered from ice samples gathered from the community, samples from 27 hospital ice machines yielded low numbers (<10 CFU/mL) of a variety of potentially opportunistic microorganisms, mainly gram-negative bacilli.⁸¹⁹ During the survey period, no healthcare-associated infections were attributed to the use of ice. Ice from community sources appeared to have higher levels of microbial contamination (75% - 95% of 194 samples had total heterotrophic plate counts <500 CFU/mL, with the proportion of positive cultures dependent on the incubation temperature) and showed evidence of fecal contamination from the source water.⁸¹⁹ Thus, ice machines in health care are no more heavily contaminated compared to ice machines in the community. If the source water for ice in a healthcare facility is not fecally-contaminated, then ice from clean ice machines and chests should pose no special hazard for immunocompetent patients. Some waterborne bacteria found in ice could potentially be a risk to immunocompromised patients if they consume ice or drink beverages with ice. For example, *Burkholderia cepacia* in ice could present an infection risk for cystic fibrosis patients.^{819, 820} It may therefore be prudent to protect immunosuppressed and otherwise medically at-risk patients from exposure to tap water and ice potentially contaminated with opportunistic pathogens.⁹

Currently there are no microbiological standards for ice, ice-making machines, or ice storage equipment, although several investigators have suggested the need for such standards.^{819, 826} Culturing of ice machines is not routinely recommended but may be useful as part of an epidemiologic investigation.^{827 - 829} Sampling might also help determine the best schedule for cleaning open ice-storage chests. Recommendations for a regular program of maintenance and disinfection have been published.^{826 - 829} Healthcare facilities are advised to clean ice-storage chests at least monthly, with more frequent cleanings recommended for open chests. Portable ice chests and containers require cleaning and low-level disinfection before the addition of ice intended for consumption. Ice-making machines may require less frequent cleaning, but their maintenance is important to proper performance (Tables 29 and 30).

Table 29. General Steps to Clean and Disinfect Ice Machines

1. Disconnect the unit from the power supply.
 2. Remove and discard the ice in the bin.
 3. Allow the unit to warm to room temperature.
 4. Clean with fresh water and detergent.
 5. Rinse with fresh tap water.
 6. Wipe dry with clean materials.
 7. Rinse with a dilute solution of chlorine bleach (10 - 100 ppm [1 - 8 mL of bleach per gallon of water]).
 8. Let all surfaces air dry before returning the unit to service.
-

Ice and ice-making machines may also be contaminated via improper storage or handling of ice by patients and/or staff.^{653 - 655, 815 - 818, 830} Suggested steps to avoid this means of contamination include: 1) minimizing or avoiding direct hand contact with ice intended for consumption; 2) using a hard-surface scoop to dispense ice; and 3) installing machines that dispense ice directly into portable containers at the touch of a control.^{654, 829}

Table 30. General Steps to Maintain Ice Machines

1. Disconnect the unit from the power supply.
 2. Remove and discard the ice.
 3. Disassemble the removable parts of the machine that make contact with the water to make ice.
 4. Thoroughly clean the machine and the parts.
 5. Check for any needed repair.
 6. Ensure the presence of an air space in the tubing that leads from the water inlet into the water distribution system of the machine.
 7. Inspect for rodent or insect infestations under the unit and treat if necessary.
 8. Check door gaskets (open compartment models) for evidence of leakage or dripping into the storage chest.
 9. Clean the ice-storage chest as described in Table 29.
 10. Sanitize the machine by circulating a 50 - 100 ppm chlorine solution through the ice-making and storage systems (suggested contact time: 4 hrs. for 50 ppm solution, 2 hrs. for 100 ppm solution).
 11. Drain the chlorine solution, and flush with fresh tap water.
 12. Allow the ice-storage chest to dry, and return to service.
-

8. Hydrotherapy Tanks and Pools

a. General Information

Hydrotherapy equipment (e.g., pools, whirlpools [jacuzzis], hot tubs, physiotherapy tanks) has traditionally been used to treat patients with medical conditions which include, but are not limited to burns,^{831, 832} septic ulcers, lesions, amputations,⁸³³ orthopedic impairments and injuries, arthritis,⁸³⁴ and more recently, kidney lithotripsy.⁶³⁰ Wound-care medicine is increasingly moving away from hydrotherapy, however, in favor of bedside pulsed-lavage therapy using sterile solutions for cleaning and irrigation.^{472, 835 - 838} Several episodes of healthcare-associated infections have been linked to use of hydrotherapy equipment (Table 31). Potential routes of infection include incidental ingestion of the water, sprays and aerosols, and direct contact with

Table 31. Infections Associated with Use of Hydrotherapy Equipment

Microorganism(s)	Medical Condition(s)	Reference(s)
<i>Acinetobacter baumannii</i>	Sepsis	550
<i>Citrobacter freundii</i>	Cellulitis	839
<i>Enterobacter cloacae</i>	Sepsis	840
<i>Legionella</i> spp.	Legionellosis	841
<i>Mycobacterium abscessus</i> , <i>M. fortuitum</i> , <i>M. marinum</i>	Skin ulcers, soft tissue infections	593 - 595, 842
<i>Pseudomonas aeruginosa</i>	Sepsis, soft tissue infections, folliculitis, wound infections	472, 473, 486, 648, 844 - 846
Adenovirus, adeno-associated virus	Conjunctivitis	847

wounds and intact skin (folliculitis). Risk factors for infection include: 1) age and sex of the patient; 2) underlying medical conditions; 3) length of time spent in the hydrotherapy water; and 4) portals of entry.⁸⁴⁸

Infection control for hydrotherapy tanks, pools, or birthing tanks presents unusual challenges because indigenous microorganisms will always be present in the water during treatments. In addition, some studies have found free living amoebae (i.e., *Naegleria lovaniensis*) which are commonly found in association with *N. fowleri* in hospital hydrotherapy pools.⁸⁴⁹ Although there are instances when patients with wounds, burns, or other types of non-intact skin conditions receive treatment in hydrotherapy equipment, it is neither practical nor warranted to consider this equipment as “semi-critical” in accordance with the Spaulding classification.⁸⁵⁰ Microbial data to evaluate the risk of infection to patients using hydrotherapy pools or birthing tanks are insufficient. Nevertheless, healthcare facilities should maintain stringent cleaning and disinfection practices in accordance with the manufacturer’s instructions and with relevant scientific literature until data supporting more rigorous infection control measures become available.

b. Hydrotherapy Tanks

Hydrotherapy tanks (e.g., whirlpools, Hubbard tanks) are shallow tanks constructed of stainless steel, Plexiglass, or tile. They are closed-cycle water systems with hydrojets to circulate, aerate, and agitate the water. The maximum water temperature range is 10°C - 40°C (50°F - 104°F). The warm water temperature, constant agitation and aeration, and design of the hydrotherapy tanks provide ideal conditions for bacterial proliferation if the equipment is not properly cleaned and maintained. Associated equipment (e.g., parallel bars, plinths, Hoyer lifts, wheelchairs) can also be potential reservoirs of microorganisms, depending on the materials used in these items (i.e., porous vs. non-porous materials) and the surfaces that may become wet during use. Patients with active skin colonizations and wound infections can serve as sources of contamination for the equipment and the water. Contamination from spilled tub water can extend to drains, floors, and walls.^{649 - 652} Healthcare-associated colonization or infection can result from exposure to endogenous sources of microorganisms (autoinoculation) or exogenous sources (via cross-contamination from other patients previously receiving treatment in the unit).

Although some facilities have used tub liners to minimize on environmental contamination of the tanks, the use of a tub liner does not eliminate the need for cleaning and disinfection. Draining these small pools and tanks after each patient use, thoroughly cleaning with a detergent, and disinfecting according to manufacturers’ instructions have been shown to reduce bacterial contamination levels in the water from 10⁴ CFU/mL to <10 CFU/mL.⁸⁵¹ The general recommendation is to maintain a chlorine residual of 15 ppm in the water prior to the patient’s therapy session (e.g., by adding 15 grams of calcium hypochlorite 70% [e.g., HTH®] per 100 gallons of water).⁸⁵¹ A study of commercial and residential whirlpools found that superchlorination or draining, cleaning, disinfection, and refilling of whirlpools markedly reduced densities of *Pseudomonas aeruginosa* in the whirlpool water.⁸⁵² The bacterial populations were rapidly replenished, however, when disinfectant concentrations dropped below recommended levels for recreational use (i.e., chlorine at 3.0 ppm or bromine at 6.0 ppm).

A few reports describe the addition of antiseptic chemicals to hydrotherapy tank water, especially for burn patient therapy.^{853 - 855} One small study showed a reduction in the number of *Pseudomonas* spp. and other gram-negative bacteria from both patients and equipment surfaces when chloramine-T (“chlorazene”) is added to the water.⁸⁵⁶ Chloramine-T has not, however, been approved for water treatment in the United States.

c. Hydrotherapy Pools

Hydrotherapy pools typically serve large numbers of patients and are usually heated to 33°C - 37°C (91.4°F - 98.6°F). The temperature range is more narrow (35°C - 36°C [95°F - 96.8°F]) for pediatric and geriatric patient use.⁸⁵⁷ Because the size of hydrotherapy pools precludes draining after patient use, proper management is required to maintain the proper balance of water conditioning (i.e., alkalinity, hardness, temperature) and disinfection. The most widely used chemicals for disinfection of pools are chlorine and chlorine compounds -- calcium hypochlorite, sodium hypochlorite, lithium hypochlorite, chloroisocyanurates, and chlorine gas. Solid and liquid formulations of chlorine chemicals are the easiest and safest to use.⁸⁵⁸ Other halogenated compounds have also been used for pool water disinfection, albeit on a limited scale. Bromine, which forms bactericidal bromamines in the presence of ammonia, has limited use because it has been associated with contact dermatitis.⁸⁵⁹ Iodine does not bleach hair, swim suits, or cause eye irritation, but when introduced at proper concentrations, it will give water a greenish-yellowish cast.⁸⁵¹

In practical terms, maintenance of large hydrotherapy pools, such as those used for exercise, is similar to that for indoor public pools (i.e., continuous filtration, chlorine residuals no less than 0.4 ppm, and pH between 7.2 -7.6).^{860, 861} Supply pipes and pumps also need to be maintained to eliminate these as possible reservoirs for waterborne organisms.⁸⁶² Specific standards for chlorine residual and pH of the water are addressed in local and state regulations. Patients who are fecally-incontinent or who have draining wounds should refrain from using these pools until their condition improves.

d. Birthing Tanks and Other Equipment

The use of birthing tanks, jacuzzis, and whirlpools is a recent addition to obstetrical practice.⁸⁶³ Few studies on the potential risks associated with these pieces of equipment have been conducted. In one small study of 32 women, a newborn contracted a *Pseudomonas* infection, the strain of which was identical to the organism isolated from the tank water.⁸⁶⁴ Other studies have shown no significant increases in the rates of post-immersion infections among mothers and infants.^{865, 866}

Because the water and the tub surfaces routinely become contaminated with the mother’s skin flora and blood during labor and delivery, birthing tanks and other tub equipment need to be drained after each patient use and the surfaces thoroughly cleaned and disinfected. Healthcare facilities are advised to follow the manufacturer’s instructions for selection of disinfection method and chemical germicide. Chlorine residuals for public whirlpools and jacuzzis ranges from 2 - 5 ppm.⁸⁶⁷ Use of an inflatable tub is an alternative solution, but this item must be cleaned and disinfected between patients if it is not considered as a single-use unit.

A recent trend in health care is to use recreational tanks or jacuzzis as hydrotherapy equipment. Although such home equipment appears to be suitable for hydrotherapy, they are neither designed nor constructed to function in this capacity. Additionally, manufacturers are generally not obligated to provide the healthcare facility with cleaning and disinfecting instructions appropriate for medical equipment use, and the U.S. Food & Drug Administration (FDA) does not evaluate recreational equipment. Healthcare facilities should therefore carefully evaluate this “off-label” use of home equipment before proceeding with a purchase.

9. Miscellaneous Medical Equipment Connected to Main Water Systems

a. Automated Endoscope Reprocessors

The automated endoscopic reprocessor (AER) is classified by the FDA as an accessory for the flexible fiberoptic endoscope.⁶³⁰ A properly operating AER can provide a more consistent, reliable method of decontaminating and terminal reprocessing for endoscopes between patient procedures than manual reprocessing methods alone.^{868, 869} An endoscope is generally subjected to high-level disinfection using a liquid chemical sterilant. The optimal rinse fluid for a disinfected endoscope would be sterile water, since the instrument is a semi-critical device.³ Sterile water, however, is expensive and difficult to produce in sufficient quantities and with adequate quality assurance for instrument rinsing in

an AER. Therefore, one option that is used for AERs is rinse water which has been passed through filters with a pore size of 0.1 - 0.2 μm (i.e., to render the water “bacteria-free”). These filters are usually located in the water line at or near the port where the mains water enters the equipment.

Two general situations have linked water to contamination of flexible fiberoptic endoscopes: 1) rinsing a disinfected endoscope with unfiltered tap water, followed by storage of the instrument without drying out the internal channels; and 2) contamination of AERs from tap water inadvertently introduced into the equipment. In the latter instance, the machine’s water reservoirs and fluid circuitry become contaminated with waterborne, heterotrophic bacteria (e.g., *Pseudomonas aeruginosa*, NTM) which can survive and persist in biofilms attached to these components.^{870 - 873} Colonization of the reservoirs and water lines of the AER becomes a problem if the required cleaning, disinfection, and maintenance are not performed on the equipment as recommended by the manufacturer.^{872 - 874} Use of the 0.1 - 0.2 μm filter in the water line helps to keep bacterial contamination to a minimum,^{869, 873, 875} but filters may fail and allow bacteria to pass on through.^{876, 877} Filters also require maintenance for proper performance.^{875, 878} Increasing attention to the proper disinfection of the connectors that hook the instrument to the AER may help to further reduce the potential for contaminating endoscopes during reprocessing.⁸⁷⁹

Studies have linked deficiencies in endoscope cleaning and/or disinfecting processes to the incidence of post-endoscopic adverse outcomes.^{880 - 883} Several clusters have been traced to AERs of older designs, and these were associated with water quality.^{870 - 872, 884} Regardless of whether manual or automated terminal reprocessing is used for endoscopes, the internal channels of the instrument must be dried before storage. The presence of residual moisture in the internal channels encourages the proliferation of waterborne microorganisms, some of which may be potentially pathogenic. Using 70% isopropyl alcohol to flush the internal channels, followed by forced air drying of these channels and hanging the endoscope vertically in a protected cabinet will ensure internal drying of the endoscope and lessen the potential for proliferation of waterborne microorganisms, whether they originate from the disinfectant water or from residual contamination from the previous patient.^{873, 874, 881, 885} This is part of the worldwide standard process for successful endoscope reprocessing.⁸⁸⁶

An additional problem with waterborne microbial contamination of AERs centers on increased microbial resistance to sterilants such as alkaline glutaraldehyde.^{874, 887} Opportunistic waterborne microorganisms (e.g., *Mycobacterium chelonae*, *Methylobacterium* spp.) have been associated with pseudo-outbreaks, colonization, and infection in clinical settings such as bronchoscopy.^{874, 887, 888} The problem of increasing microbial resistance to glutaraldehyde has been attributed to improper use of the disinfectant in the equipment which allows the dilution of glutaraldehyde to fall below the manufacturer’s recommended minimal use concentration of 1.5%.⁸⁸⁷

b. Dental Unit Water Lines

Dental unit water lines (DUWLs) consist of small-bore plastic tubing that delivers water used for general, non-surgical irrigation and as a coolant to dental handpieces, sonic and ultrasonic scalers, and air-water syringes; municipal tap water is the source water for these lines. The presence of biofilms of waterborne bacteria and fungi (e.g., *Legionella* spp., *Pseudomonas aeruginosa*, NTM) in DUWLs has been well established.^{613, 662, 663, 889, 890} Biofilms continually release planktonic microorganisms into the water, the titers of which can exceed 1×10^6 CFU/mL.⁶⁶² To date, however, scientific evidence indicates there is little risk of significant adverse health effects among immunocompetent persons due to contact with water from a dental unit. Nonetheless, exposing patients or dental personnel to water of uncertain microbiological quality is not consistent with universally accepted infection control principles.⁸⁹¹

In 1993, the CDC issued guidelines relative to water quality in a dental setting. These guidelines recommend that all dental instruments that use water (including high-speed handpieces) should be run to discharge water for 20-30 seconds after each patient and for several minutes before the start of each clinic day.⁸⁹² Although these guidelines are designed to help reduce the number of microorganisms present in treatment water, they do not address the issue of reducing or preventing biofilm formation in the waterlines.

The numbers of microorganisms in water used as coolant or irrigant for non-surgical dental treatment should be as low as reasonably achievable and, at a minimum, should meet nationally recognized standards for safe drinking water.⁸⁹³ There is minimal evidence that water that meets drinking water standards poses a health hazard for immunocompetent

persons. The EPA, the American Public Health Association (APHA), and the American Water Works Association (AWWA) have set a maximum limit for aerobic, heterotrophic, mesophilic bacteria in drinking water at 500 CFU/mL.⁸⁹⁴⁸⁹⁵ This standard is achievable today, given improvements in water line technology. An upcoming revision to the 1993 CDC dental infection control guidelines may consider a standard such as that for dialysis water quality (i.e., ≤ 200CFU/mL) be adopted for DUWLs. Dentists should consult with the manufacturer of their dental unit to determine the best equipment and method for maintaining and monitoring good water quality.⁸⁹¹

E. Environmental Services

1. Principles of Cleaning and Disinfecting Environmental Surfaces

Although microbiologically-contaminated surfaces can serve as reservoirs of potential pathogens, these surfaces are generally not directly associated with transmission of infections to either staff or patients. The transferral of microorganisms from environmental surfaces to patients is largely via hand contact with the surface.^{896, 897} While hand hygiene/handwashing is important to minimize the impact of this transferral, cleaning and disinfecting environmental surfaces as appropriate is fundamental in reducing their potential contribution to the incidence of healthcare-associated infections.

The principles of cleaning and disinfecting environmental surfaces take into account the intended use of the surface or item in patient care. CDC retains the Spaulding classification for medical and surgical instruments which outlines three categories based on the potential for the instrument to transmit infection if the instrument is microbiologically contaminated before use.^{898, 899} These categories are “critical,” “semi-critical,” and “non-critical.” In 1991, CDC proposed an additional category designated “environmental surfaces” to Spaulding’s original classification.⁹⁰⁰ These are non-critical surfaces that generally do not come into direct contact with patients during care. Environmental surfaces carry the least risk of disease transmission and can be safely decontaminated using less rigorous methods than those used on medical instruments and devices. Environmental surfaces can be further divided into medical equipment surfaces (e.g., knobs or handles on hemodialysis machines, x-ray machines, instrument carts, dental units) and housekeeping surfaces (e.g., floors, walls, tabletops).⁹⁰⁰

Several factors influence the choice of disinfection procedure for environmental surfaces: 1) the nature of the item to be disinfected; 2) the number of microorganisms present; 3) the innate resistance of those microorganisms to the inactivating effects of the germicide; 4) the amount of organic soil present; 5) the type and concentration of germicide used; 6) duration and temperature of germicide contact; and 7) if using a proprietary product, other specific indications for use.⁹⁰¹

Cleaning is the necessary first step of any sterilization or disinfection process. Cleaning is a form of decontamination that renders the environmental surface safe to handle or use by removing organic matter, salts, and visible soils, all of which interfere with microbial inactivation.^{902 - 908} The physical action of scrubbing with detergents and surfactants and rinsing with water removes large numbers of microorganisms from surfaces.⁹⁰⁵ If the surface is not cleaned before the terminal reprocessing procedures are started, then the success of the sterilization or disinfection process is compromised.

Disinfection is a generally less lethal process compared to sterilization, and usually involves the use of liquid chemical germicides (disinfectants). By definition, chemical disinfection differs from sterilization by its lack of sporicidal power; disinfection eliminates virtually all recognized pathogenic microorganisms, but not necessarily all microbial forms (e.g., bacterial spores) on inanimate surfaces. Accordingly, disinfection procedures lack the margin of safety achieved by sterilization processes, which are most frequently accomplished by physical means (e.g., heat).

Spaulding proposed three levels of disinfection for the treatment of devices and surfaces that do not require sterility for safe use. These disinfection levels are “high-level,” “intermediate-level,” and “low-level.”^{898, 899} The basis for these levels is that microorganisms can usually be grouped according to their innate resistance to a spectrum of physical or chemical germicidal agents (Table 32). This information, coupled with the instrument/surface classification, determines the appropriate level of terminal disinfection for an instrument or surface.

Table 32. Levels of Disinfection by Type of Microorganism^{2, 900}

Levels	Bacteria			Fungi ^a	Viruses	
	Vegetative	Tubercle Bacillus	Spores		Lipid & Medium Size	Nonlipid & Small
High	[^b	[[^c	[[[
Intermediate	[[[^d	[[[^e
Low	[—	—	[[[

a Includes asexual spores but not necessarily chlamydo spores or sexual spores.

b Plus sign indicates that a killing effect can be expected when the normal use-concentrations of chemical disinfectants or pasteurization are properly employed; a negative sign indicates little or no killing effect.

c Only with extended exposure times are high-level disinfectant chemicals capable of killing high numbers of bacterial spores in laboratory tests; they are, however, capable of sporicidal activity.

d Some intermediate-level disinfectants (e.g., hypochlorites) can exhibit some sporicidal activity; others (e.g., alcohols, phenolics) have no demonstrable sporicidal activity.

e Some intermediate-level disinfectants, although they are tuberculocidal, may have limited virucidal activity.

The process of high-level disinfection, an appropriate standard of treatment for heat-sensitive, semi-critical medical instruments (e.g., flexible, fiberoptic endoscopes), is capable of inactivating all vegetative bacteria, mycobacteria, viruses, fungi, and some bacterial spores if they are present. High-level disinfection is accomplished with powerful, sporicidal chemicals (e.g., glutaraldehyde, peracetic acid, and hydrogen peroxide) that are *not* appropriate for use on housekeeping surfaces. Intermediate-level disinfection does not necessarily kill bacterial spores, but does inactivate *Mycobacterium tuberculosis* var. *bovis*, which is significantly more resistant to chemical germicides than ordinary vegetative bacteria, fungi, and medium- to small viruses (with or without lipid envelopes). Chemical germicides with sufficient potency to achieve intermediate-level disinfection include but are not limited to chlorine-containing compounds (e.g., sodium hypochlorite), alcohols, some phenolics, and some iodophors. Low-level disinfection inactivates vegetative bacteria, fungi, enveloped viruses (e.g., human immunodeficiency virus [HIV], influenza viruses), and some non-enveloped viruses (e.g., adenoviruses). Low-level disinfectants, which may also be referred to as “sanitizers,” include quaternary ammonium compounds, some phenolics, and some iodophors. Germicidal chemicals cleared as skin antiseptics are not appropriate for use as environmental surface disinfectants.⁹⁰⁰

The selection and use of chemical germicides are guided by product label instructions and information.

Sterilant/disinfectant chemicals (i.e., high-level disinfectants) are regulated now exclusively by the FDA as a result of recent memoranda of understanding between FDA and the EPA which delineates agency authority for chemical germicide regulation.^{909, 910} Environmental surface germicides (i.e., intermediate- and low-level disinfectants) are regulated by the EPA and labeled with EPA registration numbers. The labels and package inserts of these germicides specify indications for product use and provide claims for the range of antimicrobial activity. The EPA requires certain pre-registration laboratory potency tests for these products to support product label claims, but does not perform these pre-registration laboratory tests, relying instead on the manufacturer to provide valid data. Germicides labeled as “hospital disinfectant” have passed the potency tests for activity against three representative microorganisms - *Pseudomonas aeruginosa*, *Staphylococcus aureus*, and *Salmonella cholerae suis*. Hospital disinfectants with demonstrated potency against mycobacteria (i.e., intermediate-level disinfectants) may list “tuberculocidal” on the label as well. Low-level disinfectants are often labelled “hospital disinfectant” without a tuberculocidal claim, because they lack the potency to inactivate mycobacteria. Other claims, such as “fungicidal,” “pseudomonicidal,” or “virucidal” may appear on labels of environmental surface germicides, but the designations of “tuberculocidal hospital disinfectant” and “hospital disinfectant” correlate directly to Spaulding’s assessment of intermediate-level disinfectants and low-level disinfectants, respectively.⁹⁰⁰

A common misconception in the use of surface disinfectants in health care relates to the underlying purpose when using proprietary products labeled as a “tuberculocidal” germicide. Such products will not interrupt and prevent the transmission of TB in healthcare settings because TB is not acquired from environmental surfaces. The tuberculocidal

claim is used as a benchmark by which to measure germicidal potency. Since mycobacteria have the highest intrinsic level of resistance among the vegetative bacteria, viruses, and fungi, any germicide with a tuberculocidal claim on the label (i.e., an intermediate-level disinfectant) is considered capable of inactivating a broad spectrum of pathogens, including much less resistant organisms such as the bloodborne pathogens (e.g., HBV, hepatitis C virus [HCV], and HIV). It is this broad spectrum capability, rather than the product's specific potency against mycobacteria, that is the basis for protocols and OSHA regulations dictating use of tuberculocidal chemicals for surface disinfection.⁹¹¹

2. General Cleaning Strategies for Patient-Care Areas

The number and types of microorganisms present on environmental surfaces are influenced by several factors: 1) number of people in the environment; 2) amount of activity; 3) amount of moisture; 4) presence of material capable of supporting microbial growth; 5) rate at which organisms suspended in the air are removed; and 6) type of surface and orientation [horizontal or vertical].⁹¹² Strategies for cleaning and disinfecting surfaces in patient-care areas take into account: 1) potential for direct patient contact; 2) degree and frequency of hand contact; and 3) potential contamination of the surface with body substances or environmental sources of microorganisms (e.g., soil, dust, or water).

a. Strategies for Routine Cleaning of Medical Equipment

Manufacturers of medical equipment should provide care and maintenance instructions specific to their equipment. These instructions should include information about materials compatibility with chemical germicides, whether or not the equipment can be safely immersed for cleaning, and how the equipment should be decontaminated if servicing is required.⁹¹¹ In the absence of manufacturers' instructions, non-critical medical equipment (e.g., stethoscopes, blood pressure cuffs, dialysis machines, equipment knobs and controls) usually need only cleaning followed by low- to intermediate-level disinfection depending on the nature and degree of contamination. Ethyl alcohol or isopropyl alcohol in concentrations ranging from 60% to 90% is often used to disinfect small surfaces (e.g., rubber stoppers of multiple-dose medication vials, thermometers)^{901, 913} and external surfaces of equipment (e.g., stethoscopes, ventilators) on occasion. However, alcohol evaporates rapidly, which makes extended contact times difficult to achieve unless items are immersed, a factor which precludes its practical use as a large surface disinfectant.⁹⁰¹ Alcohol may cause discoloration, swelling, hardening, and cracking of rubber and certain plastics after prolonged and repeated use, and damage the shellac mounting of lenses in medical equipment.⁹¹⁴

Barrier protection of surfaces and equipment is useful, especially if these surfaces are: 1) touched frequently by gloved hands during the delivery of patient care; 2) likely to become contaminated with body substances; or 3) difficult to clean. Impervious-backed paper, aluminum foil, plastic or fluid-resistant covers are suitable for use as barrier protection. An example of this approach is the use of plastic wrapping to cover the handle of the operatory light in dental care settings.⁸⁹² Coverings should be removed and discarded while the healthcare worker is still gloved.⁸⁹² The healthcare worker, after un gloving and hand hygiene, covers these surfaces with clean materials before the next patient encounter.

b. Strategies for Routine Cleaning of Housekeeping Surfaces

Housekeeping surfaces require regular cleaning and removal of soil and dust. Dry conditions favor the persistence of gram-positive cocci (e.g., coagulase-negative *Staphylococcus* spp.) in dust and on surfaces, whereas moist, soiled environments favor the growth and persistence of gram-negative bacilli.^{897, 915, 916} Fungi are also present on dust and proliferate in moist, fibrous material.

Most, if not all, housekeeping surfaces need to be cleaned only with soap and water or a detergent/disinfectant, depending on the nature of the surface and the type and degree of contamination. Cleaning and disinfection schedules and methods vary according to the area of the hospital, type of surface to be cleaned, and the amount and type of soil present. Disinfectant-detergent formulations registered by the EPA are used for environmental surface cleaning, but the actual physical removal of microorganisms and soil by scrubbing is probably as important, if not more so, than any antimicrobial effect of the cleaning agent used.⁹¹⁷ Therefore, cost, safety, and acceptability by housekeepers can be the main criteria for selecting a registered agent. If using a proprietary detergent/disinfectant, the manufacturers' instructions for appropriate use of the product should be followed. Consult the products' material safety data sheets (MSDS) to determine appropriate precautions to prevent hazardous conditions during product application.

Housekeeping surfaces can be divided into two groups - those with minimal hand-contact (e.g., floors, ceilings), and

those with frequent hand-contact (“high touch surfaces”). The methods, thoroughness, and frequency of cleaning and the products used are determined by healthcare facility policy.⁶ However, high-touch housekeeping surfaces in patient-care areas (e.g., doorknobs, bedrails, light switches, wall areas around the toilet in the patient’s room) should be cleaned and/or disinfected more frequently than surfaces with minimal hand-contact. Infection control practitioners typically use a risk-assessment approach to identify high-touch surfaces and then coordinate an appropriate cleaning and disinfecting strategy and schedule with the housekeeping staff.

Horizontal surfaces with infrequent hand contact (e.g., window sills, hard-surface flooring) in routine patient-care areas require cleaning on a regular basis, when soiling or spills occur, and when a patient is discharged.⁶ Regular cleaning of surfaces and decontamination as needed is also advocated to protect potentially exposed workers.⁹¹¹ Cleaning of walls, blinds, and window curtains is recommended when they are visibly soiled.^{916, 917} Disinfectant fogging is not recommended for general infection control in routine patient-care areas.²

Extraordinary cleaning and decontamination of floors in healthcare settings is unwarranted. Studies have demonstrated that disinfection of floors offers no significant advantage over regular detergent/water cleaning and has little or no impact on the occurrence of healthcare-associated infections.^{896, 897, 919 - 921} Additionally, newly cleaned floors become rapidly recontaminated from airborne microorganisms and those transferred from shoes, equipment wheels, and body substances.^{915, 919, 922} Methods for cleaning non-porous floors include wet mopping and wet vacuuming, dry dusting with electrostatic materials, and spray buffing.^{917, 923 - 925} Methods that produce minimal mists and aerosols or dispersion of dust in patient-care areas are preferred.^{9, 20, 109, 262}

Part of the cleaning strategy is to minimize contamination of cleaning solutions and cleaning tools. Bucket solutions become contaminated almost immediately during cleaning, and continued use of the solution transfers increasing numbers of microorganisms to each subsequent surface to be cleaned.^{915, 922, 926} Cleaning solutions should be replaced frequently. A variety of “bucket” methods have been devised to address the frequency with which cleaning solutions are replaced.^{927, 928} Another source of contamination in the cleaning process is the cleaning cloth or mop head, especially if left soaking in dirty cleaning solutions.^{915, 929 - 931} Laundering of cloths and mop heads after use, and allowing them to dry before re-use, can help to minimize the degree of contamination.⁹³¹ A simplified approach to cleaning involves replacing soiled cloths and mop heads with clean items each time a bucket of detergent/disinfectant is emptied and replaced with fresh, clean solution.⁹³² Disposable cleaning cloths and mop heads are an alternative option, if costs permit.

Another reservoir for microorganisms in the cleaning process may be dilute solutions of the detergents or disinfectants, especially if the working solution is prepared in a dirty container and stored for long periods of time. Gram-negative bacilli (e.g., *Pseudomonas* spp.) have been detected in working solutions of some disinfectants (e.g., phenolics).⁹³³ Application of contaminated cleaning solutions, particularly from small-quantity aerosol spray bottles or with equipment that might generate aerosols during operation, should be avoided, especially in high-risk patient areas.^{934, 935} Making sufficient fresh cleaning solution for daily cleaning, discarding any remaining solution, and drying out the container will help to minimize the degree of bacterial contamination. Containers (e.g., quart-sized dishwashing liquid bottles) which dispense liquid as opposed to spray-nozzle dispensers may be used to apply detergent/disinfectants to surfaces and then to cleaning cloths with minimal aerosol generation. A pre-mixed, “ready-to-use” detergent/disinfectant solution may be used if available.

c. Strategies for Cleaning Special-Care Areas

Guidelines have been published on cleaning strategies for isolation areas and for the operating rooms.^{6, 7} The basic strategies for areas housing immunosuppressed patients include: 1) wet dusting horizontal surfaces daily with cleaning cloths pre-moistened with a hospital disinfectant;^{94, 927} 2) using care when wet dusting equipment and surfaces above the patient to avoid patient contact with the detergent/disinfectant; 3) avoiding the use of cleaning equipment that produces mists or aerosols; 4) equipping vacuums with HEPA filters, especially for the exhaust, for use in any patient-care area where immunosuppressed patients;^{9, 94, 927} and 5) regular cleaning and maintenance of equipment to ensure efficient particle removal. Dispersal of microorganisms in the air from dust or aerosols can be problematic in these settings than elsewhere in the facility. There is the potential for vacuum cleaners to serve as dust disseminators if they are not operating properly.⁹³⁶ Doors to patients’ rooms should be closed when vacuuming anywhere in patient-care

areas where immunosuppressed patients are located.⁹ Bacterial and fungal contamination of filters in cleaning equipment is inevitable, and these filters should be cleaned regularly or replaced as per equipment manufacturer instructions.

Tacky mats in operating rooms and other patient-care areas do little to minimize the overall degree of contamination of floors, and have little impact on the incidence rate of healthcare-associated infection in general.^{340, 915, 924} An exception to this statement is the use of tacky mats inside the entry ways of cordoned-off construction areas inside the healthcare facility; these mats help to minimize the intrusion of dust into patient-care areas.

Special precautions for cleaning incubators, mattresses, and other nursery surfaces have been recommended to address reports of hyperbilirubinemia in newborns linked to inadequately diluted solutions of phenolics and poor ventilation.^{937 - 939} These medical conditions have not, however, been associated with the use of properly prepared use-solutions of phenolics. Non-porous housekeeping surfaces in neonatal units can be disinfected with properly-diluted or pre-mixed phenolics, followed by rinsing with clean water, and can be an option among available hospital disinfectants.⁹³⁹ Phenolics are not recommended for cleaning infant bassinets and incubators during the stay of the infant. Infants who remain in the nursery for an extended period should be moved periodically to freshly cleaned and disinfected bassinets and incubators.⁹³⁹ If phenolics are used for terminal cleaning of bassinets and incubators, the surfaces should be rinsed thoroughly with water and dried before reused of either piece of equipment. Cleaning and disinfecting protocols should allow for the full contact time specified for the product used. Bassinette mattresses should be replaced, however, if the mattress cover surface is broken.⁹³⁹

3. Cleaning Strategies for Spills of Blood and Body Substances

There is no evidence that either HBV, HCV, or HIV has ever been transmitted from a housekeeping surface (i.e., floors, walls, or countertops). Nonetheless, prompt removal and surface disinfection of an area contaminated by either blood or body substances are sound infection control practices and OSHA requirements.⁹¹¹

Studies have shown that HIV is inactivated rapidly after being exposed to commonly used chemical germicides at concentrations that are much lower than those used in practice.^{940 - 945} HBV is readily inactivated with a variety of germicides, including quaternary ammonium compounds.⁹⁴⁶ Embalming fluids (e.g., formaldehyde) are also capable of completely inactivating HIV and HBV.^{947, 948} In addition to commercially available germicides registered for use as “hospital disinfectants” with a tuberculocidal claim (i.e., intermediate-level disinfectants), a solution of sodium hypochlorite (household chlorine bleach) prepared daily is an inexpensive and effective broad-spectrum germicide. Concentrations of sodium hypochlorite solutions ranging from approximately 5,000 ppm (1:10 dilution of household bleach) to 500 ppm (1:100 dilution) free chlorine are effective depending on the amount of organic material (e.g., blood, mucus, urine) present on the surface to be cleaned and disinfected.^{949, 950} Commercially available chemical germicides may be more compatible with certain materials that might be corroded by repeated exposure to sodium hypochlorite, especially the 1:10 dilution. Appropriate personal protective equipment (e.g., gloves, goggles) should be worn when preparing hypochlorite solutions.⁹¹¹

Strategies for decontaminating spills of blood and other body fluids differ based on the setting in which they occur and the volume of the spill.⁹⁴⁹ In patient-care areas, workers can manage small spills with a one-step procedure.^{927, 928} For spills containing large amounts of blood or other body substances, workers should first remove visible organic matter with absorbent material (e.g., disposable paper towels discarded into leak-proof, properly labeled containment) and then clean and decontaminate the area.^{944, 945, 951} If the surface is nonporous and the germicide of choice is household bleach, then a 1:100 dilution is appropriate for the decontamination step. This assumes, however, that: 1) the worker assigned to clean the spill is wearing gloves and other personal protective equipment appropriate to the task; 2) the majority of the organic matter of the spill has been removed with absorbent material; and 3) the surface has been cleaned to remove residual organic matter. A recent study showed that even strong chlorine solutions (i.e., 1:10 dilution of chlorine bleach) may fail to totally inactivate high titers of virus in large quantities of blood, but in the absence of blood these disinfectants can achieve complete viral inactivation.⁹⁵⁰ This supports the need to remove the majority of organic matter from a large spill before final disinfection of the surface. Additionally, EPA-registered proprietary disinfectant label claims are based on use on a pre-cleaned surface.^{900, 902}

Managing spills of blood, body fluids, or other infectious materials in clinical, public health, and research laboratories requires more stringent measures because of the higher potential risk of disease transmission associated with large volumes of blood and body fluids, and high numbers of microorganisms associated with diagnostic cultures. The use of an intermediate-level germicide for routine decontamination in the laboratory is prudent.⁹⁰² Recommended practices for managing large spills of concentrated infectious agents in the laboratory include: 1) confining the contaminated area; 2) flooding the area with a liquid chemical germicide before cleaning; and 3) decontaminating with fresh germicidal chemical of at least intermediate-level disinfectant potency.⁹⁴⁹ A suggested technique when flooding the spill with germicide is to lay absorbent material down on the spill and apply sufficient germicide to thoroughly wet both the spill and the absorbent material.⁹⁵² If using a solution of household chlorine bleach, a 1:10 dilution is recommended for this purpose. Commercial germicides should be used according to the manufacturers' instructions for use dilution and contact time. Gloves should be worn during the cleaning and decontamination procedures in both clinical and laboratory settings. Personal protective equipment in such a situation may include the use of respiratory protection (e.g., N95 respirator) if clean-up procedures are expected to generate infectious aerosols. Protocols for cleaning spills should be developed and on record as part of good laboratory practice.⁹⁵²

4. Carpeting and Cloth Furnishings

a. Carpeting

Carpeting has been used for over 30 years in both public and patient-care areas of healthcare facilities. Advantages of carpeting in patient-care areas include: 1) its noise-limiting characteristics; 2) the "humanizing" effect on health care; and 3) its contribution to reductions in falls and resultant injuries, particularly for the elderly.^{953 - 955} Compared to hard-surface flooring, however, carpeting is harder to keep clean, especially after spills of blood and body substances. It is also harder to push equipment with wheels on carpeting (e.g., wheelchairs, carts, gurneys).

Several studies have documented the presence of diverse microbial populations, primarily bacteria and fungi, in carpeting.^{111, 956 - 963} The variety and number of microorganisms tend to be stable over. New carpeting quickly becomes colonized, with bacterial growth plateauing after about four weeks.⁹⁵⁸ Additionally, vacuuming and cleaning the carpeting can temporarily reduce the numbers of bacteria, but these populations soon rebound and return to pre-cleaning levels.^{958, 959, 962} Bacterial contamination tends to increase with higher levels of activity.^{957 - 959, 964} Soiled carpeting that is or remains damp or wet provides an ideal setting for the proliferation and persistence of gram-negative bacteria and fungi. Carpeting that remains in this condition should be removed.

Despite the evidence of bacterial growth and persistence in carpeting, there is little epidemiologic evidence to show that carpets influence healthcare-associated infection rates in areas housing *immunocompetent* patients.^{962, 964} This guideline, therefore, includes no recommendations against the use of carpeting in these areas. Nonetheless, it is reasonable to avoid the use of carpeting in areas where spills are likely to occur (e.g., laboratories, areas around sinks, janitor closets) and where patients may be at greater risk of infection from airborne environmental pathogens (e.g., HSCT units, burn units, intensive care units, operating rooms).^{111, 966} An outbreak of aspergillosis in an HSCT unit was recently attributed to carpet contamination and a particular method of carpet cleaning.¹¹¹ A window in the unit had been opened repeatedly during the time of a nearby building fire, which allowed fungal spore intrusion into the unit. After the window was sealed, the carpeting was cleaned using a "bonnet buffing" machine, which dispersed *Aspergillus* spores into the air.¹¹¹ Wet vacuuming was instituted, replacing the dry cleaning method used previously; no additional cases of invasive aspergillosis were identified.

The care setting and the method of carpet cleaning are important factors to consider in efforts to minimize or prevent production of aerosols and dispersal of carpet microorganisms into the air.^{94, 111} Both vacuuming and shampooing or wet cleaning with equipment can disperse microorganisms to the air.^{111, 936} Vacuum cleaners should be maintained to minimize dust dispersal in general, and be equipped with HEPA filters, especially for use in high-risk patient-care areas.^{9, 94, 927} Some formulations of carpet-cleaning chemicals, if applied or used improperly, can be dispersed into the air as a fine dust capable of causing respiratory irritation in patients and staff.⁹⁶⁷ Cleaning equipment, especially those that do wet cleaning and extraction, can become contaminated with waterborne organisms (e.g., *Pseudomonas aeruginosa*) and serve as a reservoir for these organisms if this equipment is not properly maintained. Use of such equipment then may transfer large numbers of bacteria to carpeting during the cleaning process.⁹⁶⁸ It is, therefore, important to keep the carpet cleaning equipment in good repair, and to allow the unit to dry out between uses if so indicated by the

manufacturer.

General carpet cleaning should be performed on a regular basis determined by internal policy, although spills of blood and body substances require prompt spot cleaning using standard cleaning procedures and application of chemical germicides.⁹¹¹ Most, if not all, modern carpet brands suitable for public facilities are able to tolerate the activity of a variety of liquid chemical germicides. However, OSHA considers that, compared to nonporous floor surfaces, carpeting contaminated with blood or other potentially infectious materials cannot be fully decontaminated.⁹⁶⁹ To comply with the intent of this interpretation, facilities electing to use carpeting for high-activity patient-care areas may choose carpet tiles in areas at high risk for spills.^{911, 969} In the event of contamination with blood or other body substances, carpet tiles can be removed, discarded, and replaced.

Over the last few years, some carpet manufacturers have treated their products with fungicidal and/or bacteriocidal chemicals. Although these chemicals may help to reduce the overall numbers of bacteria or fungi present in carpet, their use does not preclude the routine care and maintenance of the carpeting. Limited evidence suggests that chemically treated carpet may have helped to keep healthcare-associated aspergillosis rates low in one HSCT unit,¹¹¹ but overall there is no indication that use of treated carpeting will prevent the incidence of healthcare-associated infections in care areas for immunocompetent patients.

b. Cloth Furnishings

Upholstered furniture and furnishings are becoming increasingly common in patient-care areas. These furnishings range from simple cloth chairs in patients' rooms to a complete decorating scheme that gives the interior of the facility more the look of an elegant hotel.⁹⁷⁰ Even though pathogenic microorganisms have been isolated from the surfaces of cloth chairs, there is no epidemiologic evidence that general patient-care areas with cloth furniture have increased rates of healthcare-associated infection compared to areas with hard-surfaced furniture.^{971, 972} Allergens, such as dog or cat dander, have been detected in or on cloth seating in clinics and elsewhere in hospitals in concentrations higher than that found on bed linens.^{973, 974} These are presumably transferred from the clothing of visitors. Researchers have therefore suggested that cloth chairs should be vacuumed regularly to keep the dust and allergen levels to a minimum. This recommendation, however, has generated concerns that aerosols created from vacuuming could place immunocompromised patients or patients with preexisting lung disease (e.g., asthma) at risk for development of healthcare-associated, environmental airborne disease.^{9, 20, 109, 929} At present it is reasonable to minimize the use of upholstered furniture and furnishings in any patient-care areas where immunosuppressed patients are located (e.g., HSCT units).⁹

5. Flowers and Plants in Patient-Care Areas

Fresh flowers, dried flowers, and potted plants are common items in healthcare facilities. In 1974, clinicians isolated an *Erwinia* sp. post mortem from a neonate diagnosed with fulminant septicemia, meningitis, and respiratory distress syndrome.⁹⁷⁵ Since *Erwinia* spp. are plant pathogens, plants brought into the delivery room were suspected as the source of the bacteria, although the case report did not definitively establish a direct link. A number of subsequent studies evaluated the numbers and diversity of microorganisms in the vase water of cut flowers. These studies revealed that high concentrations of bacteria, ranging from 10⁴ - 10¹⁰ CFU/mL, were often present, especially if the water was changed infrequently.^{495, 670, 976} The major group of microorganisms in flower vase water was gram-negative bacteria, with *Pseudomonas aeruginosa* the most frequently isolated organism.^{495, 670, 976, 977} *P. aeruginosa* was also the major organism directly isolated from chrysanthemums and other potted plants.^{978, 979} However, flowers in hospitals were not significantly more contaminated with bacteria compared to flowers in restaurants or in the home.⁶⁷⁰ Additionally, there were no differences in the diversity and degree of antibiotic resistance of bacteria isolated from hospital flowers compared to bacteria from flowers elsewhere.⁶⁷⁰

Despite the diversity and large numbers of bacteria associated with flower vase water and potted plants, there is little or no evidence to indicate that the presence of plants in *immunocompetent* patient-care areas poses an increased risk of healthcare-associated infection.⁴⁹⁵ In one small study among surgical patients, no correlation was observed between bacterial isolates from flowers in the area with the incidence and etiology of postoperative infections among the patients.⁹⁷⁷ Similar conclusions were reached in a study which looked at the bacteria of potted plants.⁹⁷⁹ Nonetheless, it

is prudent to implement some precautions for general patient-care settings, such as: 1) limiting flower and plant care to staff with no direct patient contact; 2) if this is not feasible, then advising healthcare staff to wear gloves when handling plants; 3) washing hands after handling plants; 4) changing vase water every two days and discharging the water into a sink outside the immediate patient environment; and 5) cleaning and disinfecting vases after use.⁶⁷⁰

Some researchers have looked into the possibility of adding a chemical germicide to vase water to control bacterial populations. Chemicals such as hydrogen peroxide and chlorhexidine appear to be reasonably well tolerated by plants.^{977, 980, 981} Use of these chemicals, however, was not evaluated in studies to assess impact on healthcare-associated infection rates. Modern florists now have a variety of products available to add to vase water to extend the life of cut flowers and to minimize bacterial clouding of the water.

Flowers (fresh and dried) and ornamental plants, however, may serve as a reservoir of *Aspergillus* spp., and dispersal of conidiospores into the air from this source is a strong possibility.¹⁰⁹ Healthcare-associated outbreaks of invasive aspergillosis reinforce the importance of maintaining an environment as free of *Aspergillus* spp. spores as possible for patients with severe, prolonged neutropenia. Both fresh-cut flowers and dried flower arrangements may provide a reservoir for these fungi as well as other fungal species (e.g., *Fusarium* spp.).^{109, 982} Researchers in one of the small studies of bacteria and flowers suggested that flowers and vase water should be avoided in areas providing care to medically at-risk patients (e.g., oncology patients, transplant patients), although this study did not attempt to correlate the observations of bacterial populations in the vase water with the incidence of healthcare-associated infections.⁴⁹⁵ It is therefore reasonable to exclude flowers and plants from areas where immunosuppressed patients may be located (e.g., HSCT units).⁹

6. Pest Control

Cockroaches, flies and maggots, ants, mosquitos, spiders, mites, midges, and mice are among the typical arthropod and vertebrate pest populations found in healthcare facilities. Insects can serve as agents for the mechanical transmission of microorganisms, or as active participants in the disease transmission process by serving as a vector.^{983 - 985} Arthropods recovered from healthcare facilities have been shown to carry a wide variety of pathogenic microorganisms.^{986 - 992} Studies have suggested that the diversity of microorganisms associated with insects reflects the microbial populations present in the indoor healthcare environment; some pathogens encountered in insects from hospitals were either absent from or present to a lesser degree in insects trapped from residential settings.^{993 - 996} Some of the microbial populations associated with insects in hospitals have demonstrated resistance to antibiotics.^{984, 995, 997, 998}

Insect habitats are characterized by warmth, moisture, and availability of food.⁹⁹⁹ Insects forage in and feed on substrates, including but not limited to food scraps from kitchens/cafeteria, foods in vending machines, discharges on dressings either in use or discarded, other forms of human detritus, medical wastes, human wastes, and routine solid waste.^{993 - 997} Cockroaches, in particular, have been known to feed on fixed sputum smears in laboratories.^{1000, 1001} Both cockroaches and ants are frequently found in the laundry, central sterile supply departments, or anywhere in the facility where water or moisture is present (e.g., sink traps, drains, janitor closets). Ants will often find their way into sterile packs of items as they forage in a warm, moist environment.⁹⁹³ Cockroaches and other insects frequent loading docks and other areas with direct access to the outdoors.

Although insects carry a wide variety of pathogenic microorganisms on their surfaces and in their gut, the direct association of insects with disease transmission (apart from vector transmission) is largely circumstantial, especially in healthcare settings; insects do not appear to play a major or singular role in healthcare-associated disease transmission in developed countries. Some studies have been conducted to examine the role of houseflies as possible vectors for shigellosis and other forms of diarrheal disease in non-healthcare settings.^{983, 1002} When control measures aimed at reducing the fly population density were implemented, a concomitant reduction in the incidence of diarrheal infections, carriage of *Shigella* organisms, and mortality due to diarrhea among infants and young children were observed.

From a public health and hygiene perspective, it is reasonable to control and eradicate arthropod and vertebrate pests from all indoor environments, including healthcare facilities.^{1003, 1004} Modern approaches to institutional pest management usually focus on: 1) eliminating food sources, indoor habitats, and other conditions that attract pests; 2) excluding pests from the indoor environments; and 3) applying pesticides as needed.¹⁰⁰⁵ Sealing windows in modern

healthcare facilities helps to minimize insect intrusion. When windows need to be opened for ventilation, ensuring that screens are in good repair and closing doors to the outside can help with pest control. Insects need to be kept out of all areas of the healthcare facility, but this is especially important for the operating rooms and any area where immunosuppressed patients are located. A pest control specialist with appropriate credentials can provide a regular insect control program that is tailored to the needs of the facility and uses approved chemicals and/or physical methods. Industrial hygienists can provide information on possible adverse reactions of patients and staff to pesticides and suggest alternative methods for pest control as needed.

7. Special Pathogen Concerns

a. Antibiotic-Resistant Gram-Positive Cocci

Vancomycin-resistant enterococci (VRE), methicillin-resistant *Staphylococcus aureus* (MRSA), and *S. aureus* with intermediate levels of resistance to glycopeptide antibiotics (VISA or GISA) represent serious and increasing concerns for infection control. While the term GISA is technically a more accurate description of the strains isolated to date, most of which are classified as having intermediate resistance to both vancomycin and teicoplanin, the term “glycopeptide” may not be recognized by many clinicians. Thus the label of VISA, which emphasizes a change in minimum inhibitory concentration (MICs) to vancomycin, is similar to that of VRE and is more meaningful to clinicians.¹⁰⁰⁶ According to National Nosocomial Infection Surveillance (NNIS) statistics for infections acquired among intensive care unit patients in the United States in 1999, 52.3% of infections due to *S. aureus* were identified as MRSA infections, and 25.2% of enterococcal infections were attributed to VRE. These figures reflect a 37% and a 43% increase, respectively, since 1994-98.¹⁰⁰⁷

People represent the major reservoir of *S. aureus*.¹⁰⁰⁸ Although *S. aureus* has been isolated from a variety of environmental surfaces (e.g., stethoscopes, floors, charts, furniture, dry mops, hydrotherapy tanks), the role of environmental contamination in transmission of this organism appears to be minimal.^{1009 - 1012} *S. aureus* contamination of surfaces and tanks within burn therapy units, however, may be important in the transmission of infection among burn patients.¹⁰¹³

The colonized patient is the principal reservoir of VRE, and patients who are immunosuppressed (e.g., transplant patients) or otherwise medically at-risk (e.g., intensive care unit patients, cardio-thoracic surgical patients, patients previously hospitalized for extended periods, or those having received multi-antimicrobial or vancomycin therapy) appear to be at greatest risk for VRE colonization.^{1014 - 1017} The mechanisms by which cross-colonization take place are not well defined, although recent studies have indicated that both MRSA and VRE may be transmitted either: 1) directly from patient to patient; 2) indirectly by transient carriage on the hands of healthcare workers;^{1018 - 1021} or 3) by hand transfer of these gram-positive organisms from contaminated environmental surfaces and patient-care equipment.^{1014, 1017, 1022 - 1027} In one survey, hand carriage of VRE in workers in a long-term care facility ranged from 13 - 41%.¹⁰²⁸ Many of the environmental surfaces found to be contaminated with VRE in outbreak investigations have been those which are touched frequently by the patient or the healthcare worker.¹⁰²⁹ Such high-touch surfaces include, but are not limited to bedrails, doorknobs, bed linens, gowns, overbed tables, blood pressure cuffs, computer table, bedside tables, and various medical equipment.^{22, 1017, 1024, 1025, 1030} Contamination of environmental surfaces with VRE generally occurs in areas where colonized patients are present,^{1017, 1022, 1024, 1025} but the potential for contamination increases when such patients have diarrhea,¹⁰¹⁷ or have multiple body site colonization.¹⁰³¹ Additional factors which can be important in the dispersion of these pathogens to environmental surfaces are misuse of glove techniques by healthcare workers, especially when cleaning fecal contamination from surfaces; and patient, family, and visitor hand hygiene and personal hygiene.

Interest in the importance of environmental reservoirs of VRE increased when laboratory studies demonstrated that enterococci can persist in a viable state on dry environmental surfaces for extended periods of time (7 days to 4 months)^{1029, 1032} and multiple strains can be identified during extensive periods of surveillance.¹⁰³¹ VRE can be recovered from inoculated hands of healthcare workers (with or without gloves) for up to 60 minutes.²² The presence of either MRSA, VISA, or VRE on environmental surfaces, however, does not mean that patients in the contaminated areas will become colonized. Strict adherence to handwashing and the proper use of barrier precautions help to minimize the potential for spread of these pathogens. Published recommendations for preventing the spread of vancomycin resistance address isolation measures, including patient cohorting and management of patient-care items.⁵

Careful cleaning of patient rooms and medical equipment is important to the overall control of MRSA, VISA, or VRE transmission, but should not be the major focus of a control program for either VRE or MRSA. Routine cleaning and disinfection of the housekeeping surfaces (e.g., floors, walls) and patient-care surfaces (e.g., bedrails) should be adequate for inactivation of these organisms. Both MRSA and VRE are susceptible to a variety of low- and intermediate-level disinfectants such as alcohols and sodium hypochlorite; quaternary ammonium compounds, phenolics, and iodophors at recommended use dilutions for environmental surface disinfection.^{1033 - 1036} Additionally, both VRE and vancomycin-sensitive enterococci appear to be equally sensitive to inactivation by chemical germicides,^{1033, 1034, 1036} and similar observations have been made when comparing the germicidal resistance of MRSA to that of either methicillin-sensitive *S. aureus* (MSSA) or VISA.¹⁰³⁷ There is no indication for using stronger solutions of disinfectants for inactivation of either VRE, MRSA, or VISA because of the organisms' resistance to antibiotics.^{1037, 1038} VRE from clinical specimens have exhibited some measure of increased tolerance to heat inactivation in temperature ranges <100°C (<212°F),^{1033, 1039} but the clinical significance of these observations is unclear because the role of cleaning the surface or item prior to heat treatment wasn't evaluated. Although routine environmental sampling is not recommended, laboratory surveillance of environmental surfaces during episodes when VRE contamination is suspected can help determine the effectiveness of the cleaning and disinfecting procedures. Environmental culturing should be approved and supervised by the infection control program in collaboration with the clinical laboratory.^{1014, 1017, 1018, 1022, 1026}

One concern is that standard procedures during terminal cleaning and disinfection of surfaces may be inadequate for the elimination of VRE from patient rooms.^{1039 - 1042} Given the sensitivity of VRE to hospital disinfectants, current disinfecting protocols should be effective if they are diligently carried out and properly performed. Healthcare facilities should be sure that housekeeping staff use correct procedures for cleaning and disinfecting surfaces in VRE-contaminated areas. These include using sufficient amounts of germicide at proper use dilution and adequate contact time of the surface with the germicide liquid.¹⁰⁴²

b. *Clostridium difficile*

Clostridium difficile is the most frequent etiologic agent for healthcare-associated diarrhea.^{1043, 1044} In one hospital, 30% of adults who developed healthcare-associated diarrhea were positive for *C. difficile*.¹⁰⁴⁵ Most patients remain asymptomatic after infection, but the organism continues to be shed in their stools. Risk factors for acquiring *C. difficile*-associated infection include: 1) exposure to antibiotic therapy, particularly with β -lactam agents;¹⁰⁴⁶ 2) gastrointestinal procedures and surgery;¹⁰⁴⁷ 3) advanced age; and 4) indiscriminate use of antibiotics.^{1048 - 1051} Of all the measures that have been used to prevent the spread of *C. difficile*-associated diarrhea, the most successful measure has been the restriction of the use of antimicrobial agents.^{1052, 1053}

Clostridium difficile is an anaerobic, gram-positive bacterium. Normally fastidious in its vegetative state, it is capable of sporulating when environmental conditions no longer support its continued growth. The capacity to form spores enables the organism to persist in the environment (e.g., in soil, on dry surfaces) for extended periods of time. Environmental contamination by this microorganism is well known, especially wherever fecal contamination may occur.¹⁰⁵⁴ There is little evidence, especially for housekeeping surfaces (e.g., floors, walls), that the environment is a direct source of infection for patients.^{963, 1055 - 1059} However, direct exposure to contaminated patient-care items (i.e., rectal thermometers) and high-touch surfaces in patients' bathrooms have been implicated as sources of infection.^{1053, 1058, 1060, 1061}

Transfer of the pathogen to the patient via the hands of healthcare workers is thought to be the most likely mechanism of exposure.^{24, 1056, 1062} Standard isolation techniques intended to minimize enteric contamination of patients, healthcare worker hands, patient-care items, and environmental surfaces have been published.⁹²⁶ Handwashing remains the major means of reducing hand contamination. Proper use of gloves is an ancillary measure that helps to further minimize transfer of these pathogens from one surface to another.

The degree to which the environment becomes contaminated with *C. difficile* spores is proportional to the number of patients with *C. difficile*-associated diarrhea,^{24, 1055, 1058} although asymptomatic, colonized patients may also serve as a source of contamination. Few studies have examined the use of specific chemical germicides for the inactivation of *C. difficile* spores, and no well-controlled trials to determine efficacy of surface disinfection and its impact on healthcare-associated diarrhea have been conducted. Some investigators have evaluated the use of chlorine-containing chemicals

(e.g., 1:100 dilutions of unbuffered hypochlorite, phosphate-buffered hypochlorite [1600 ppm]), and showed that the number of contaminated environmental sites was reduced by half.¹⁰⁵⁸ The recommended approach to environmental infection control with respect to *C. difficile* is meticulous cleaning and disinfection using proper use dilutions and contact times for environmental surface germicides as appropriate.^{901, 1053, 1063}

c. Respiratory and Enteric Viruses in Pediatric Care Settings

Although the viruses mentioned here are not unique to the pediatric care setting in healthcare facilities, their prevalence in these areas, especially during the winter months, is significant. Children, particularly neonates, are more likely to develop infection with significant clinical disease from these agents compared to adults, and accordingly are more likely to require supportive care during their illness.

Common respiratory viruses in pediatric care areas include rhinoviruses, respiratory syncytial virus (RSV), adenoviruses, influenza viruses, and parainfluenza viruses. Transmission of these viruses occurs primarily via direct contact with small-particle aerosols or via hand contamination with respiratory secretions that are then transferred to the nose or eyes. Since transmission primarily requires close personal contact, contact precautions are appropriate to interrupt transmission.⁶ Hand contamination can occur from direct contact with secretions, or indirectly from touching high-touch environmental surfaces that have become contaminated with virus from large droplets. The efficiency of the latter form of transmission is dependent on the ability of these viruses to survive on environmental surfaces. Infectious RSV has been recovered from skin, porous surfaces, and non-porous surfaces after 30 minutes, 1 hour, and 7 hours respectively.¹⁰⁶⁴ Parainfluenza viruses are known to persist for up to 4 hours on porous surfaces and up to 10 hours on non-porous surfaces.¹⁰⁶⁵ Rhinoviruses can persist on porous surfaces and non-porous surfaces for approximately 1 and 3 hours respectively; study participants in a controlled environment became infected with rhinoviruses after first touching a surface with dried secretions and then touching their nasal or conjunctival mucosa.¹⁰⁶⁶ Although the efficiency of direct transmission of these viruses from surfaces in uncontrolled settings remains to be defined, these data underscore the basis for maintaining regular protocols for cleaning and disinfecting of high-touch surfaces.

The clinically important enteric viruses encountered in pediatric care settings include enteric adenovirus, astroviruses, caliciviruses, and rotavirus. Group A rotavirus is the most common cause of infectious diarrhea in infants and children; transmission is primarily fecal-oral. The role of fecally-contaminated surfaces and fomites in rotavirus transmission is unclear. During one epidemiologic investigation of enteric disease among children attending day-care, rotavirus contamination was detected on 19% of inanimate objects in the center.^{1067, 1068} In an outbreak in a pediatric unit, secondary cases of rotavirus infection tended to cluster in areas where children with rotaviral diarrhea were located.¹⁰⁶⁹ Outbreaks of small round-structured viruses (i.e., caliciviruses [Norwalk virus and Norwalk-like viruses]) can affect both patients and staff, with attack rates $\geq 50\%$.¹⁰⁷⁰ Routes of person-to-person transmission include fecal-oral spread and aerosols from vomiting.^{1071 - 1073} Fecal contamination of surfaces in care settings may potentially spread large amounts of either virus to the environment. Studies which have attempted to use low- and intermediate-level disinfectants to inactivate rotavirus suspended in feces have demonstrated the protective effect of high concentrations of organic matter.^{1074, 1075} Intermediate-level disinfectants (e.g., alcoholic quaternary ammonium compounds, chlorine solutions) can be effective in inactivating enteric viruses provided that a cleaning step to remove most of the organic matter precedes terminal disinfection.¹⁰⁷⁵ These findings underscore the need for proper cleaning and disinfecting procedures where contamination of environmental surfaces with body substances is likely. Using disposable, protective barrier coverings may help to minimize the degree of surface contamination.⁸⁹²

d. Creutzfeldt-Jakob Disease (CJD) in Patient-Care Areas

Creutzfeldt-Jakob disease (CJD) is a rare, invariably fatal, transmissible spongiform encephalopathy (TSE) occurs worldwide with an average annual incidence of 1 case per million population.^{1076 - 1078} CJD is one of several TSEs affecting humans; others include kuru, fatal familial insomnia, and Gerstmann-Sträussler-Scheinker syndrome. A TSE that affects a younger population (compared to the age range of CJD cases) has been described primarily in the United Kingdom since 1996.¹⁰⁷⁹ This variant form of CJD (vCJD) is clinically and neuropathologically distinguishable from classic CJD, and there is strong epidemiologic and laboratory evidence which suggests a causal association between bovine spongiform encephalopathy (BSE [Mad Cow disease]) and vCJD.^{1079 - 1082}

The agent associated with CJD is a prion, which is an abnormal isoform of a normal protein constituent of the central

nervous system.^{1083 - 1085} The mechanism by which the normal form of the protein is converted to the abnormal, disease-causing prion is unknown. The tertiary conformation of the abnormal prion protein appears to confer a heightened degree of resistance to conventional methods of sterilization and disinfection.^{1086, 1087}

Although the majority of classic CJD cases (~ 90%) occur sporadically, an extremely limited number of cases are the result of a direct exposure to prion-containing material, usually central nervous system tissue, or pituitary hormones. Designated iatrogenic cases, these have been linked to pituitary hormone therapy,^{1088 - 1090} transplants of either dura mater or corneas,^{1091 - 1097} or neurosurgical instruments and depth electrodes.^{1098 - 1101} In the cases involving instruments and depth electrodes, inadequate cleaning and terminal reprocessing of these devices failed to fully inactivate the contaminating prions.

Prion inactivation studies involving whole tissues and tissue homogenates have been conducted to determine the parameters of physical and chemical methods of sterilization or disinfection necessary for complete inactivation.^{1086, 1102 - 1107} The application of these findings to environmental infection control in healthcare settings is problematic. Despite a consensus that abnormal prions display some extreme measure of resistance to inactivation by either physical or chemical methods, scientists disagree about the exact conditions needed for sterilization. Inactivation studies utilizing whole tissues present extraordinary challenges to any sterilizing method.¹¹⁰⁸ Additionally, the experimental design of these studies preclude the evaluation of surface cleaning as a part of the total approach to pathogen inactivation.¹¹⁰⁸

Some researchers have recommended the use of 1:2 to full-strength sodium hypochlorite (20,000 - 50,000 ppm) or 1-2 N sodium hydroxide (NaOH) for the inactivation of prions on surfaces, such as in the pathology laboratory.^{1086, 1104} Although this may be appropriate for the decontamination of laboratory, operating room, or autopsy room surfaces with central nervous system tissue contact from a known or suspected patient, this approach is not indicated for routine or terminal cleaning of a room previously occupied by a CJD patient. Both solutions pose hazards for the healthcare worker doing the decontamination. NaOH is caustic and should not make contact with the skin. Sodium hypochlorite solutions (chlorine bleach) can corrode metals such as aluminum. MSDS information should be consulted when attempting to work with concentrated solutions of either chemical.

Environmental infection control strategies need to be based on the principles of the “Chain of Infection,” regardless of the disease of concern.¹³ Although CJD is transmissible, it is not highly contagious. To date, all iatrogenic cases of CJD have been linked to a direct exposure to prion-contaminated central nervous system tissue or pituitary hormones. The six documented iatrogenic cases associated with instruments and devices involved neurosurgical instruments and devices that introduced residual contamination directly to the recipient’s brain. There is no evidence to date that vCJD has been transmitted iatrogenically. There is no evidence that either CJD or vCJD has been transmitted from environmental surfaces such as the housekeeping surfaces. Therefore, routine procedures are adequate for terminal cleaning and disinfection of a CJD patient’s room. Additionally, epidemiologic studies on highly transfused patients indicate that blood does not appear to be an source for prion transmission.^{1109 - 1114} Routine procedures for containing, decontaminating, and disinfecting surfaces with blood spills should be adequate for proper infection control in these situations. Guidance for environmental infection control in operating rooms and autopsy areas has been published.^{1113, 1114} Disposable, impermeable coverings should be used during autopsies to minimize surface contamination. Surfaces that have become contaminated with central nervous system tissue or cerebral spinal fluid should be cleaned and decontaminated by: 1) removing the majority of the tissue or body substance with absorbent materials; 2) wetting the surface with a sodium hypochlorite solution containing minimally $\geq 20,000$ ppm (e.g., a 1:2 dilution at a minimum) or a 1 - 2 N NaOH solution for 1- 2 hours; and 3) rinsing thoroughly.^{1113, 1114}

F. Environmental Sampling

(Note: This portion of Part I will address basic principles and methods of sampling environmental surfaces and sources primarily for microorganisms, whereas the applied strategies of sampling with respect to environmental infection control has been discussed previously in the appropriate subsections).

1. General Principles - Microbiologic Sampling of the Environment

Before 1970, U.S. hospitals conducted regularly scheduled culturing of the air and environmental surfaces such as floors,

walls, and table tops.¹¹¹⁵ By 1970, CDC and the American Hospital Association (AHA) were advocating the discontinuation of routine environmental culturing because rates of healthcare-associated infection had not been related to levels of general microbial contamination of air or environmental surfaces, and meaningful standards for permissible levels of microbial contamination of environmental surfaces or air did not exist.^{1116 - 1118} Between 1970 and 1975, 25% of U.S. hospitals reduced the extent of such routine environmental culturing; this trend has continued since then.^{1119, 1120}

Random, undirected sampling (referred to as “routine” in previous guidelines) differs from the current practice of targeted sampling for defined purposes.^{2, 1117} Previous recommendations against routine sampling were not intended to discourage the use of sampling for which sample collection, culture, and interpretation are conducted in accordance with defined protocols.² In this guideline, targeted microbiological sampling connotes a monitoring process that includes: 1) a written, defined, multidisciplinary protocol for sample collection and culturing; 2) analysis and interpretation of results using scientifically determined or anticipatory baseline values for comparison; and 3) expected actions based on the results obtained.

Microbiological sampling of air, water, and inanimate surfaces (i.e., environmental sampling) is an expensive and time-consuming process which is complicated by many variables in protocol, analysis, and interpretation. It is therefore indicated for only four situations.¹¹²¹ The first is to support of an investigation of an outbreak of disease or infections when environmental reservoirs or fomites are implicated epidemiologically in disease transmission.^{159, 1122, 1123} It is important that such culturing be supported by epidemiologic data. Environmental sampling, as with all laboratory testing, should not be conducted if there is no plan for interpreting and acting on the results obtained.^{11, 1124, 1125} Linking microorganisms from environmental samples with clinical isolates by molecular epidemiology is crucial.

The second situation for which environmental sampling may be warranted is in research. Well-designed and controlled experimental methods and approaches can provide new information about the spread of healthcare-associated diseases.^{126, 129} A classic example is the study of environmental microbial contamination that compared healthcare-associated infection rates in an old hospital and a new facility before and shortly after occupancy.⁸⁹⁶

The third indication for sampling is to monitor a potentially hazardous environmental condition, confirm the presence of a hazardous chemical or biological agent, and validate the successful abatement of the hazard. This type of sampling can be used to: 1) detect bioaerosols released from the operation of healthcare equipment [e.g., an ultrasonic cleaner] and determine the success of repairs in containing the hazard;¹¹²⁶ 2) detect the release of an agent of bioterrorism in an indoor environmental setting, and determine its successful removal or inactivation; and 3) sample for industrial hygiene or safety purposes (e.g., monitor a “sick building”).

The fourth indication is for quality assurance to evaluate the effects of a change in infection control practice or ensure that equipment or systems perform according to specifications and expected outcomes. Any sampling for quality assurance purposes must follow sound sampling protocols and address confounding factors through the use of properly selected controls. Results from a single environmental sample are difficult to interpret in the absence of a frame of reference or perspective. Evaluations of a change in infection control practice are based on the assumption is that the effect will be measured over a finite period, usually of short duration. In general conducting quality assurance sampling on an extended basis, especially in the absence of an adverse outcome, is unjustified. A possible exception to this statement might be the use of air sampling during major construction periods to qualitatively detect breaks in environmental infection control measures. In one study, which began as part of an investigation of an outbreak of healthcare-associated aspergillosis, airborne concentrations of *Aspergillus* spores were measured in efforts to evaluate the effectiveness of sealing hospital doors and windows during a period of construction of a nearby building.⁵⁰ Other examples of sampling for quality assurance purposes may include commissioning newly constructed space in special care areas (i.e., operating rooms, units for immunosuppressed patients) or assessing a change in housekeeping practice. With respect to the second of the instances mentioned above, the only routine environmental microbiologic sampling generally recommended as part of a quality assurance program is the biological monitoring of sterilization processes by using bacterial spores,¹¹²⁷ and monthly cultures of water used in hemodialysis applications and for the final dialysate use dilution. Some experts also advocate periodic environmental sampling to evaluate the microbial/particulate quality for regular maintenance of the air handling system (e.g., filters) and to verify that the components of the system meet manufacturer’s specifications.²⁵⁰ Certain equipment in healthcare settings (e.g., biological safety cabinets), may also be

monitored with air flow and or particulate sampling for performance or as part of compliance with a certification program, comparing the results to a predetermined standard of performance. These measurements, however, do not normally involve microbiologic testing.

2. Air Sampling

Biological contaminants occur in the air as aerosols, and may include bacteria, fungi, viruses, and pollens.^{1128, 1129} Aerosols are characterized as solid or liquid particles suspended in air. Talking for five minutes and coughing each can produce 3,000 droplet nuclei; sneezing can generate approximately 40,000 droplets which then evaporate to particles in the size range of 0.5 - 12 μm .^{137, 1130} Particles in a biological aerosol usually vary in size from $<1 \mu\text{m}$ to $\geq 50 \mu\text{m}$. These particles may consist of a single, unattached organism or may occur in the form of clumps composed of a number of bacteria. Clumps can also include dust and dried organic or inorganic material. Vegetative forms of bacterial cells and viruses are probably present in the air in a lesser number than bacterial spores or fungal spores. Factors that determine the survival of microorganisms within a bioaerosol include: 1) the suspending medium; 2) temperature; 3) relative humidity; 4) oxygen sensitivity; and 5) exposure to UV or electromagnetic radiation.¹¹²⁸ Many vegetative cells ordinarily will not survive very long in the air unless the relative humidity and other factors are favorable for survival and the organism is enclosed within some protective cover (e.g., dried organic or inorganic matter).¹¹²⁹ Pathogens which resist drying (e.g., *Staphylococcus* spp., *Streptococcus* spp., fungal spores) will survive for relatively long periods and can be carried considerable distances while still viable. They may also settle on surfaces and become airborne again as secondary aerosols during activities such as sweeping and bed making.^{1128, 1131}

Microbiologic air sampling is used as needed to determine the numbers and types of microorganisms, or particulates in indoor air.²⁷⁸ Air sampling for quality control is, however, problematic due to lack of uniform air quality standards. Although airborne spores of *Aspergillus* spp. can pose a risk for neutropenic patients, the critical number (i.e., action level) of these spores above which outbreaks of aspergillosis would be expected to occur has not been characterized. Healthcare professionals considering the use of air sampling should keep in mind that the results represent indoor air quality at singular points of time and may be affected by a variety of factors including: 1) indoor traffic; 2) visitors coming into the facility; 3) temperature; 4) time of day or year; 5) relative humidity; 6) relative concentration of particles or organisms; and 7) the performance of the air handling system components. Air sampling results need to be compared to determinations from other defined areas, conditions, or time periods in order to be meaningful.

Table 33 summarizes the preliminary concerns when designing a microbiologic air sampling strategy. Because the amount of particulate material and bacteria retained in the respiratory system is largely dependent on the size of the inhaled particles, some thought should be given to a determination of particle size when studying airborne microorganisms and their relation to respiratory infections. Particles $>5 \mu\text{m}$ are efficiently trapped in the upper respiratory tract and are removed primarily by ciliary action.¹¹³² Particles $\leq 5 \mu\text{m}$ in diameter reach the lung, but the greatest retention in the alveoli is of particles 1-2 μm in diameter.^{1133 - 1135}

Table 33. Preliminary Concerns for Conducting Air Sampling

-
- Consider the possible characteristics and conditions of the aerosol, including size range of particles, relative amount of inert material, concentration of microorganisms, and environmental factors.
 - Determine the type of sampler, sampling time, and duration of the sampling program.
 - Determine the number of samples to be taken.
 - Ensure that adequate equipment and supplies are available.
 - Determine the method of assay which will insure optimal recovery of microorganisms.
 - Select a laboratory which will provide proper microbiologic support.
 - Ensure that samples can be refrigerated if they cannot be assayed in the laboratory immediately.
-

Bacteria, fungi, and particulates in air can be determined with basically the same methods and equipment (Table 34). The basic methods include: 1) impingement in liquids; 2) impaction on solid surfaces; 3) sedimentation; 4) filtration; 5)

centrifugation; 6) electrostatic precipitation; and 7) thermal precipitation.¹¹³¹ Of these, impingement in liquids, impaction on solid surfaces, and sedimentation (settle plates) have been used for various air sampling purposes in healthcare settings.²⁷⁸

Several instruments are available for sampling airborne bacteria and fungi. Some of the samplers are self-contained units requiring only a power supply and the appropriate collecting medium, but most require additional auxiliary equipment such as a vacuum pump and an airflow measuring device (i.e., a flowmeter or anemometer). Sedimentation or depositional methods use settle plates and therefore need no special instruments or equipment. Selection of a sampler for air sampling requires a clear understanding of the type of information desired and the particular determinations that must be made. Information may be needed on: 1) one particular organism or all organisms that may be present in the air; 2) the concentration of viable particles or of viable organisms; 3) the change of concentration with time; and/or 4) the size distribution of the collected particles. Before sampling begins, decisions as to whether the results are to be qualitative or quantitative should be made.

Table 34. Air Sampling Methods and Equipment^{278, 1131, 1136, 1137}

Method	Principle	Suited For Measuring:	Collection Media or Surface	Rate (L/min.)	Auxiliary Equipment (a)	Points to Consider	Prototype Sampler (b)
Impingement in liquids	Air drawn through small jet and directed against a liquid surface	Viable organisms; concentration over time. Example use: sampling water aerosols for <i>Legionella</i> spp.	Buffered gelatin, tryptose saline, peptone, nutrient broth	12.5	YES	Antifoaming agent may be needed. Ambient temperature and humidity will influence length of collection time.	Chemical Corps. All Glass Impinger (AGI)
Impaction on solid surfaces	Air drawn into the sampler; particles deposited on dry surface	Viable particles; viable organisms (on non-nutrient surfaces, limited to organisms that resist drying and spores); size measurements; concentration over time. Example use: sampling air for <i>Aspergillus</i> spp., fungal spores	Dry surface, coated surfaces, agar	28 (sieve) 30 - 800 (slit)	YES	Available as sieve impactors or slit impactors. Sieve impactors can be set up to measure particle size. Slit impactors have a rotating support stage for agar plates to allow for measurements of concentration over time.	Andersen Air Sampler (sieve impactor); TDL, Cassella MK-2 (slit impactors)
Sedimentation	Particles and micro-organisms settle onto surfaces via gravity	Viable particles. Example uses: sampling air for bacteria in the vicinity of and during a medical procedure; general measurements of microbial air quality.	Nutrient media (agars) on plates or slides	-	NO	Simple and inexpensive; best suited for qualitative sampling. Significant airborne fungal spores too buoyant to settle efficiently with this method.	Settle plates
Filtration	Air drawn through a filter unit; particles trapped; ~0.2 µm pore size	Viable particles; viable organisms (on non-nutrient surfaces, limited to spores and organisms which resist drying); concentration over time. Example use: air sampling for <i>Aspergillus</i> spp., fungal spores, dust	Paper, cellulose, glass wool, gelatin foam, membrane filters	1 - 50	YES	Filter must be agitated first in rinse fluid to remove and disperse trapped micro-organisms. Assay the rinse fluid. Used more for determinations of dust and chemicals.	-

(Continued next page)

Table 34 (continued). Air Sampling Methods

Method	Principle	Suited For Measuring:	Collection Media or Surface	Rate (L/min.)	Auxiliary Equipment (a)	Points to Consider	Prototype Sampler (b)
Centrifugation	Aerosols subjected to centrifugal force; particles impacted onto a solid surface	Viable particles; viable organisms (on non-nutrient surfaces, limited to spores and organisms which resist drying); concentration over time. Example use: air sampling for <i>Aspergillus</i> spp., fungal spores	Coated glass or plastic slides, agar surfaces	40 - 50	YES	Calibration is difficult and is done only by the factory. Relative comparisons of airborne contamination is its general use .	Biotest RCS Plus
Electrostatic precipitation	Air drawn over an electrostatically-charged surface; particles become charged	Viable particles; viable organisms (on non-nutrient surfaces, limited to spores and organisms resistant to drying); concentration over time	Solid collecting surfaces (glass, agar)	85	YES	High volume sampling rate, but equipment is complex and must be handled carefully. Not practical for use in healthcare settings	-
Thermal precipitation	Air drawn over a thermal gradient; particles repelled from hot surfaces, settle on colder surfaces	Size measurements	Glass coverslip, electron microscope grid	0.003 - 0.4	YES	Determines particle size by direct observation. Not commonly used because of complex adjustments and low sampling rates.	-

a Most samplers require a flow meter or anemometer and a vacuum source as auxiliary equipment.

b Trade names listed are for identification purposes only and are not intended as endorsements by the U.S. Public Health Service.

Factors to be considered when selecting a sampler are listed in Table 35.

Table 35. Factors in Selecting an Air Sampling Device¹¹³¹

-
- Viability and type of the organism to be sampled
 - Compatibility with the selected method of analysis
 - Sensitivity of particles to sampling
 - Assumed concentrations and particle size
 - Do airborne clumps need to be broken up? (i.e, total viable organism count vs. particle count)
 - Volume of air to be sampled and length of time sampler is to be continuously operated
 - Background contamination
 - Ambient conditions
 - Sampler collection efficiency
 - Effort and skill required to operate sampler
 - Availability and cost of sampler, plus back-up samplers in case of malfunction
 - Availability of auxiliary equipment and facilities, (e.g., vacuum pumps, electricity, and water).
-

Liquid impinger and solid impactor samplers are the most practical for sampling bacteria, particles, and fungal spores because they can sample large volumes of air in relatively short periods of time.²⁷⁸ Solid impactor units are available as

either “slit” or “sieve” designs. Slit impactors use a rotating disc as support for the collecting surface which allows determinations of concentration over time. Sieve impactors commonly use stages with calibrated holes of different diameters. Some impactor-type samplers use centrifugal force to impact particles onto agar surfaces. The interior of either device must be made sterile to avoid inadvertent contamination from the sampler. Results obtained from either sampling device can be expressed as organisms or particles per unit volume of air (CFU/m³).

Sampling for bacteria requires special attention because bacteria may be present as individual organisms, as clumps, or mixed with or adhering to dust or covered with a protective coating of dried organic or inorganic substances. Reports of bacterial concentrations determined by air sampling therefore must indicate whether the results represent individual organisms or particles bearing multiple cells. Certain types of samplers (e.g., liquid impingers) will completely or partially disintegrate clumps and large particles; the sampling result will therefore reflect the total number of individual organisms present in the air.

The task of sizing a bioaerosol is simplified through the use of samplers such as sieve or slit impactors because these samplers will separate the particles and microorganisms into size ranges as the sample is collected. These samplers must, however, be calibrated first by sampling aerosols under similar use conditions.¹¹³⁸

The use of settle plates (sedimentation or depositional method) is not generally recommended when sampling air for fungal spores since single spores can remain suspended in air indefinitely.²⁷⁸ Settle plates have been used mainly to sample for particulates and bacteria, either in research studies or during epidemiologic investigations.^{159, 1139 - 1142} Results of sedimentation sampling are typically expressed as numbers of viable particles or viable bacteria per unit area per the duration of sampling time (i.e., CFU/area/time); the method cannot quantify the volume of air sampled. Since the survival of microorganisms during air sampling is inversely proportional to the velocity at which the air is taken into the sampler,¹¹²⁸ one advantage of using a settle plate is its reliance on gravity to bring organisms and particles into contact with its surface, thus enhancing the potential for optimal survival of collected organisms. This process, however, takes several hours to complete, and may be impractical for all situations.

Air samplers are designed to meet differing measurement requirements. Some samplers are better suited for one form of measurement than others. No one type of sampler and assay procedure exists which can be used to collect and enumerate 100 % of airborne organisms. The sampler and/or sampling method chosen should, however, have an adequate sampling rate to collect a sufficient number of particles in a reasonable time period so that a representative sample of air is obtained for biological analysis. Newer analytical techniques for assaying air samples include PCR methods and enzyme-linked immunosorbent assays (ELISAs).

3. Water Sampling

A detailed discussion of the principles and practices of water sampling has been published.⁸⁹⁵ Water sampling in healthcare settings is used as needed to detect waterborne pathogens of healthcare concern or to determine the quality of finished water in a facility’s distribution system. Routine testing of the water in a healthcare facility is usually not indicated, but sampling in support of outbreak investigations can help determine appropriate infection control measures. Water quality assessments in dialysis settings have been discussed previously.

Healthcare facilities that determine a need for water sampling should have their samples assayed in a laboratory that uses established methods and quality assurance protocols. Water specimens are not “static specimens” at ambient temperature; potential changes in both numbers and types of microbial populations can occur during transport. Consequently, water samples should be sent to the testing laboratory cold, and testing should be done as soon as practical after collection (preferably within 24 hours).

Since most water sampling in healthcare facilities involves the testing of finished water from the facility’s distribution system, a reducing agent (i.e., sodium thiosulfate [Na₂S₂O₃]) needs to be added to neutralize residual chlorine or other halogen in the collected sample. If the water contains elevated levels of heavy metals, then a chelating agent should be added to the specimen. The minimum volume of water to be collected should be sufficient to complete any and all assays indicated; 100 mL is considered a suitable minimum volume. Sterile collection equipment should always be used.

Sampling from a tap requires flushing of the water line before sample collection. If the tap is a mixing faucet, attachments (e.g., screen, aerator) must be removed, and hot and then cold water must be run through the tap before collecting the sample.⁸⁹⁵ If the cleanliness of the tap is questionable, disinfection with 500 ppm sodium hypochlorite (1:100 dilution of chlorine bleach) and flushing the tap should precede sample collection.

Microorganisms in finished or treated water are often physically damaged (“stressed”) to the point that growth is limited when assayed under standard conditions. Such situations lead to false negative readings and misleading assessments of water quality. Appropriate neutralization of halogens and chelation of heavy metals is especially important to the recovery of these organisms. The choice of recovery media and incubation conditions will also affect the assay. Incubation temperatures should be closer to the ambient temperature of the water rather than at 37°C (98.6°F), and recovery media should be formulated to provide appropriate concentrations of nutrients to support organisms exhibiting less than rigorous growth.⁸⁹⁵ High-nutrient content media (e.g., blood agar, tryptic soy agar [TSA]) may actually inhibit the growth of these damaged organisms. Reduced nutrient media (e.g., diluted peptone, R2A) are preferable for recovery of these organisms.⁸⁹⁵

Use of aerobic, heterotrophic plate counts allows both a qualitative and quantitative measurement for water quality. If bacterial counts in water are expected to be high in number (e.g., during waterborne outbreak investigations), then assaying small quantities using pour plates or spread plates is appropriate.⁸⁹⁵ Membrane filtration is used when low-count specimens are expected and larger sampling volumes are required (≥ 100 mL). The sample is filtered through the membrane, and the filter is applied directly face-up onto the surface of the agar plate and incubated.

Unlike the testing of potable water supplies for coliforms (which uses standardized test and specimen collection parameters and conditions), water sampling to support epidemiologic investigations of disease outbreaks may be subjected to modifications dictated by the circumstances present in the facility. Assay methods for waterborne pathogens may also not be standardized. It is important, therefore, to include control or comparison samples in the experimental design. Any departure from a standard method should be fully documented and should be considered when interpreting results and developing strategies. In general, assay methods specific for waterborne pathogens of healthcare concern (e.g., *Legionella* spp., *Aeromonas* spp, *Pseudomonas* spp., *Acinetobacter* spp.) are more complicated and costly compared to methods to detect coliforms and other standard indicators of water quality.

4. Environmental Surface Sampling

Routine environmental surface sampling (e.g., surveillance cultures) in healthcare settings is neither cost-effective nor warranted.^{1138, 1143} When indicated, surface sampling should be conducted with multidisciplinary approval in adherence to carefully considered plans of action and policy; items that should be discussed prior to undertaking any sampling process are summarized in Table 36.

Table 36. Considerations before Undertaking Environmental Surface Sampling¹¹²⁷

-
- Background information from the literature and present activities [i.e., preliminary results from an epidemiologic investigation]
 - Location of surfaces to be sampled
 - The method of sample collection and the appropriate equipment for this task
 - The number of replicate samples needed and which control or comparison samples are required
 - The parameters of the sample assay method and whether the sampling will be qualitative, quantitative, or both
 - An estimate of the maximum allowable microbial numbers or types on the surface[s] sampled [refer to the Spaulding classification for devices and surfaces]
 - Some anticipation of a corrective action plan.
-

Surface sampling is currently use for research, as part of an epidemiologic investigation, or as part of a comprehensive approach for specific quality assurance purposes. As a research tool, surface sampling has been used to determine: 1) potential environmental reservoirs of pathogens;^{542, 1144 - 1146} 2) survival of microorganisms on surfaces;^{1146, 1147} or 3) the sources of the environmental contamination.⁹⁶² Some or all of these approaches can also be used during outbreak

investigations.¹¹⁴⁶ Discussion of surface sampling of medical devices and instruments is beyond the scope of this document and is deferred to future guidelines on sterilization and disinfection issues.

Meaningful results depend on the selection of appropriate sampling and assay techniques.¹¹²⁷ The media, reagents, and equipment required for surface sampling are usually available from any well-equipped microbiology laboratory and laboratory supplier. For quantitative assessment of surface organisms, non-selective, nutrient-rich agar media and broth (e.g., TSA, brain-heart infusion broth [BHI] with or without 5% sheep or rabbit blood supplement) are used for the recovery of aerobic bacteria. Broth media are used with membrane filtration techniques. Further sample work-up may require the use of selective media for the isolation and enumeration of specific groups of microorganisms. Examples of selective media are MacConkey agar (MAC [selects for gram-negative bacteria]), Cetrimide agar (selects for *Pseudomonas aeruginosa*), or Sabouraud dextrose- and malt extract agars and broths (select for fungi). Qualitative determinations of organisms from surfaces requires only the use of selective or non-selective broth media.

Effective sampling of surfaces requires moisture, either already present on the surface to be sampled or via moistened swabs, sponges, wipes, agar surfaces, or membrane filters.^{1127, 1148 - 1150} Dilution fluids and rinse fluids include various buffers or general purpose broth media (Table 37). If disinfectant residuals are expected on surfaces being sampled, then the use of specific neutralizer chemicals in both the growth media and the dilution or rinse fluids. Lists of the

Table 37. Examples of Eluents and Diluents for Environmental Surface Sampling^{1127, 1151}

<i>Solution</i>	<i>Concentration in Water</i>
Ringer	1/4 strength
Peptone water	0.1% - 1.0%
Buffered peptone water	0.067 M phosphate, 0.43% NaCl, 0.1% peptone
Phosphate-buffered saline	0.02 M phosphate, 0.9% NaCl
Sodium chloride (NaCl)	0.25% - 0.9%
Calgon Ringer ^a	1/4 strength
Thiosulfate Ringer ^b	1/4 strength
Water	
Tryptic soy broth [TSB]	
Brain-heart infusion broth [BHI] supplemented with 0.5% beef extract	

a For dissolution of calcium alginate swabs.

b For neutralization of residual chlorine

Note: A surfactant such as polysorbate (Tween® 80) may be added to eluents and diluents. A concentration in the range of 0.01% - 0.1% is generally used, depending on the specific application. Foaming may occur.

neutralizers, the target disinfectant active ingredients, and the use concentrations have already been published.^{1127, 1152} Alternatively, instead of adding neutralizing chemicals to existing culture media, or if the chemical nature of the disinfectant residuals is unknown, then the use of either commercially-available media including a variety of specific and non-specific neutralizers, or even double-strength broth media will facilitate optimal recovery of microorganisms. The inclusion of appropriate control specimens should be included to rule out both residual antimicrobial activity from surface disinfectants and potential toxicity due to the presence of neutralizer chemicals carried over into the assay system.¹¹²⁷

Methods for collecting environmental surface samples are summarized in Table 38. Specific step-by-step discussions of each of the methods have been published.^{1127, 1153} For best results, all methods should incorporate aseptic techniques, sterile equipment, and sterile recovery media.

Table 38. Methods of Environmental Surface Sampling

Method	Suitable for Use On:	Assay Technique	Procedural Notes	Points of Interpretation	Standards Available?	References
Sample/Rinse Ž Moistened swab/rinse	Ž Nonabsorbent surfaces, corners, crevices, devices, instruments	Ž Dilutions; qualitative or quantitative assays	Ž Assay multiple measured areas or devices with a single swab	Ž Report results per measured area or if assaying an object, per the entire sample site	Ž YES - food industry; NO - health care	Ž 1127, 1153 - 1156
Ž Moistened sponge/rinse	Ž Large areas, housekeeping surfaces (e.g., floors, walls)	Ž Dilutions; qualitative or quantitative assays	Ž Vigorously rub a sterile sponge over the surface	Ž Report results per measured area	Ž YES - food industry; NO - health care	Ž 1127, 1153 - 1156
Ž Moistened wipe/rinse	Ž Large areas, housekeeping surfaces (e.g., countertops)	Ž Dilutions; qualitative or quantitative assays	Ž Use a sterile wipe	Ž Report results per measured area	Ž YES - food industry; NO - health care	Ž 1127, 1153 - 1156
Direct Immersion	Small items capable of being immersed	Dilutions; qualitative or quantitative assays	Remove the item from the rinsate before assay. Use membrane filtration if rinse volume is large.	Report results per item	NO	1127
Containment	Interior surfaces of containers, tubes, bottles, etc.	Dilutions; qualitative or quantitative assays	Use membrane filtration if rinsate volume is large	Evaluate both the type(s) and numbers of microorganisms	YES - food / industrial applications for containers prior to fill	1127
RODAC*	Previously cleaned and sanitized flat, nonabsorbent surfaces. Not suitable for irregular surfaces.	Direct assay	Overgrowth results if used on heavily contaminated surfaces. Use neutralizers in the agar if surface disinfectant residuals are present.	Provides direct, quantitative results; minimum 15 plates per average hospital room	NO	1127, 1150, 1153, 1157, 1158

* RODAC = Replicate Organism Direct Agar Contact

Sample/rinse methods are frequently chosen because of their versatility. However, these sampling methods are the most prone to errors caused by manipulation of the swab, gauze pad, or sponge.¹¹⁵¹ Additionally, there should be no microbiocidal or microbiostatic agents present in any of these items when used for sampling.¹¹⁵¹ Each of the rinse methods requires effective elution of microorganisms from the item used to sample the surface. Thorough mixing of the rinse fluids after elution (e.g., via manual or mechanical mixing using a vortex mixer, shaking with or without glass beads, or ultrasonic bath) will help to remove and suspend material from the sampling device and break up clumps of organisms for a more accurate count.¹¹⁵¹ In some instances, the item used to sample the surface (e.g., gauze pad, sponge) may be immersed in the rinse fluids in a sterile bag and subjected to stomaching.¹¹⁵¹ This technique, however, is suitable only for soft or absorbent items so that the bag is not punctured during the elution process.

If sampling is conducted as part of an epidemiologic investigation of a disease outbreak, identification of isolates to species level is mandatory, and characterization beyond the species level is preferred.¹¹²⁷ When interpreting the results of the sampling, it is important to consider the expected degree of microbial contamination associated with the various categories of surfaces in the Spaulding classification. Environmental surfaces should be visibly clean; recognized pathogens in numbers sufficient to theoretically contribute to secondary transfer to other animate or inanimate surfaces should be absent from the surface being sampled.¹¹²⁷ Although the interpretation of a sample with positive microbial growth is self-evident, an environmental surface sample showing no growth does not represent a “sterile” surface, especially where housekeeping surfaces are concerned. Sensitivities of the sampling and assay methods (i.e., level of

detection) must be taken into account when no-growth samples are encountered. Properly collected control samples will help to rule out extraneous contamination of the surface sample.

G. Laundry and Bedding

1. General Information

Laundry in a healthcare facility consists of bedsheets and blankets, towels, personal clothing, uniforms, scrub suits, gowns, and drapes for surgical procedures.¹¹⁵⁹ Although soiled textiles and fabrics in healthcare facilities can be a source of large numbers of pathogenic microorganisms, reports of healthcare-associated diseases linked to soiled fabrics are so few in number that the overall risk of disease transmission during the laundry process appears to be negligible. When the incidence of such events are evaluated in the context of the volume of items laundered in healthcare settings (estimated to be 5 billion pounds annually in the United States),¹¹⁶⁰ it is apparent that existing control measures are effective in reducing the risk of disease transmission to patients and staff. Therefore, it is reasonable to encourage the continued use of current control measures to minimize the contribution of soiled laundry to the incidence of healthcare-associated infections. The control measures described here are based on principles of hygiene and common sense and pertain to laundry services utilized by healthcare facilities, either in-house or contract, rather than to laundry done in the home.

2. Epidemiology and General Aspects of Infection Control

Soiled textiles and fabrics often contain high numbers of microorganisms from body substances including, but not limited to blood, skin, stool, urine, vomitus, and other body tissues and fluids. When linen is heavily contaminated with potentially infective body substances, it can contain bacterial loads of 10^6 - 10^8 CFU/100 cm² of fabric.¹¹⁶¹ Infectious disease transmission attributed to healthcare laundry involved soiled fabrics which were handled inappropriately (i.e., shaking soiled linens). Bacteria (*Salmonella* spp., *Bacillus cereus*), viruses (hepatitis B virus), fungi (*Microsporum canis*), and ectoparasites (scabies) presumably have been transmitted from soiled textiles and fabrics to workers via either direct contact or aerosols consisting of contaminated lint generated from sorting and handling soiled linen.^{1162 - 1166} Case investigations could not, however, rule out the possibility that some of these reported infections were acquired from community sources. Through a combination of soil removal, pathogen removal, and pathogen inactivation, contaminated laundry can be rendered hygienically clean. Hygienically clean laundry carries negligible risk to healthcare workers and patients, provided that the clean textiles, fabric, and clothing are not inadvertently contaminated before use.

OSHA defines contaminated laundry as “laundry which has been soiled with blood or other potentially infectious materials or may contain sharps.”⁹¹¹ The purpose of the laundry portion of the standard is to protect the worker from exposure to potentially infectious materials during collection, handling, and sorting of soiled linens, fabrics, and textiles through the use of personal protective equipment, proper work practices, containment, labeling, hazard communication, and ergonomics.

The issue as to whether healthcare workers should be obligated to take work clothing home for laundering is complicated. OSHA regulation prohibit home laundering of items that are considered personal protective equipment (e.g., laboratory coats, surgical attire).⁹¹¹ There is disagreement, however, about whether this extends to uniforms and scrub suits which are not contaminated with blood or other potentially infectious material. Healthcare facility policies on this matter vary greatly. Uniforms without blood or body substance contamination presumably do not differ appreciably from street clothes in the degree and microbial nature of soilage. Home laundering would be expected to remove this level of soil adequately. However, if healthcare facilities require the use of uniforms, it would seem reasonable that they provide workers with clean uniforms. Healthcare facilities should address both the need to provide this service or not and to determine the frequency for laundering these items. In a recent study examining the microbial contamination of medical students' white coats, the students perceived the coats as “clean” as long as the garments were not visibly contaminated with body substances, even after wearing the coats for several weeks.¹¹⁶⁷ The heaviest bacterial load was found on the sleeves and the pockets of these garments, and the organisms most frequently isolated were *Staphylococcus aureus*, diphtheroids, and *Acinetobacter* spp.¹¹⁶⁷ The presumption here is that the sleeves of the coat may make contact with a patient and potentially serve to transfer environmentally-stable microorganisms among patients. The study, however, did not conduct surveillance among patients to detect new infections or colonizations. The students did, however, report that they would likely replace their coats more frequently and regularly if clean coats were provided.¹¹⁶⁷

Laundry services for healthcare facilities are provided either in-house or by off-site commercial laundries. The laundry facility in a healthcare setting should be designed for efficiency in providing hygienically clean textiles, fabrics, and apparel for patients and staff. Guidelines for laundry construction and operation for healthcare facilities have been published.^{120, 1168} A laundry facility is usually partitioned into two separate areas - a “dirty” area for receiving and handling the soiled laundry and a “clean” area for processing the washed items.¹¹⁶⁹ To minimize the potential for recontaminating cleaned laundry with aerosolized contaminated lint, areas receiving soiled linens should be at negative air pressure relative to the clean areas.^{1170 - 1172} Laundry areas should have handwashing facilities readily available to workers. Laundry workers should wear appropriate personal protective equipment (e.g., gloves, protective garments) while sorting soiled fabrics and textiles.⁹¹¹ Laundry equipment should be used and maintained according to the manufacturer’s instructions to prevent microbial contamination of the system.^{1164, 1173} Damp linens should not be left in machines overnight.¹¹⁶⁴

3. Collecting, Transporting, and Sorting Soiled Textiles and Fabrics

The laundry process starts with the removal of used or soiled textiles, fabrics, and/or clothing from the areas where such items are generated, including but not limited to patients’ rooms, surgical/operating areas, and laboratories. Handling soiled laundry with a minimum of agitation can help prevent the generation of potentially contaminated lint aerosols in patient-care areas.^{911, 1169} Sorting or rinsing soiled laundry at the point of generation should not be done. Soiled textiles and fabrics are placed into bags or other appropriate containment at the point of generation and securely tied or otherwise closed to prevent leakage.⁹¹¹ Single bags of sufficient tensile strength are adequate for containing laundry,¹¹⁷⁴ but leak-resistant containment is needed if the laundry is wet and can soak through a cloth bag. Bags containing soiled laundry must be clearly identified with labels, color-coding, or other methods so that healthcare workers may handle these items safely, regardless of whether the laundry is transported within the facility or destined for transport to an off-site laundry service.⁹¹¹

In the past, soiled laundry coming from isolation areas of the hospital was segregated and handled with special practices, even though few, if any, cases of healthcare-associated infection could be linked to this source.¹¹⁷⁵ Single-blinded studies have shown that laundry from isolation areas is no more heavily contaminated with microorganisms than laundry from elsewhere in the hospital.¹¹⁷⁶ Adherence to standard precautions when handling soiled laundry in isolation areas and minimizing agitation of the soiled items is considered sufficient to prevent the dispersal of potentially infectious aerosols.⁶

Soiled textiles and fabrics in bags can be transported by cart or chute.^{1168, 1172} Laundry chutes require proper design, maintenance, and use since the piston-like action of a laundry bag traveling in the chute can propel airborne microbial contaminants throughout the facility.^{1177 - 1179} Loose, soiled pieces of laundry should not be tossed into chutes.¹¹⁸⁰ Healthcare facilities should determine at what point in the laundry process textiles and fabrics should be sorted. Sorting laundry before washing protects both the machinery and fabrics from hard objects (e.g., needles, syringes, patients’ property) and reduces the potential for recontamination of clean linen.¹¹⁸¹ Sorting after washing minimizes the exposure of laundry workers to infective material in soiled fabrics and reduces airborne microbial contamination in the laundry area.¹¹⁸² Protective apparel for the workers and appropriate ventilation can minimize these exposures.^{911, 1168 - 1170}

Gloves

Gloves used for the task of sorting laundry should be of sufficient thickness to minimize sharps injuries.⁹¹¹ Employee safety personnel or industrial hygienists can help to determine the appropriate glove choice.

4. Parameters of the Laundry Process

Fabrics, textiles, and clothing used in healthcare are disinfected during laundering and generally rendered free of vegetative pathogens (hygienically clean), but they are not sterile.¹¹⁸³ Washing machines in healthcare facilities can be either washer/extractor units or continuous batch machines. A typical washing cycle consists of three main phases - a prewash, a main wash, and the rinse cycle. Cleaned wet textiles, fabrics, and clothing are then dried, pressed as needed, and prepared (e.g., folding and packaging) for distribution back to the facility. Clean linens provided by an off-site laundry must be wrapped prior to transport to prevent inadvertent contamination from dust and dirt during loading, delivery, and unloading. The antimicrobial action of the laundering process results from a combination of physical and chemical factors.^{1182, 1184, 1185} Dilution and agitation in water remove significant quantities of microorganisms. Soaps and detergents loosen soil and also have some microbicidal properties. Hot water provides an effective means of destroying

microorganisms.¹¹⁸⁶ A temperature of at least 71°C (160°F) for a minimum of 25 minutes is commonly recommended for hot-water washing.² Water of this temperature can be provided by steam jet or separate booster heater.¹²⁰ Chlorine bleach provides an extra margin of safety.^{1187, 1188} A total available chlorine residual of 50-150 ppm is usually achieved during the bleach cycle.¹¹⁸⁶ The last action in the washing process is the addition of a mild acid to neutralize any alkalinity in the water supply, soap, or detergent. The rapid shift in pH from approximately 12 to 5 may also inactivate some microorganisms.¹¹⁶¹

Chlorine bleach is an economical, broad-spectrum chemical germicide that enhances the effectiveness of the laundering process. Chlorine bleach is not, however, an appropriate laundry additive for all fabrics. Bleach was not recommended in the past for laundering flame-retardant fabrics, linens, and clothing because its use diminished the flame-retardant properties of the treated fabric.¹¹⁸³ Some modern-day flame retardant fabrics can now tolerate chlorine bleach, and chlorine alternatives such as activated oxygen-based laundry detergents provide added benefits for fabric and color safety in addition to antimicrobial activity. Oxygen-based bleach and detergents used in healthcare settings should be registered by the EPA to ensure adequate disinfection of laundry. Healthcare workers should note the cleaning instructions of textiles, fabrics, drapes, and clothing to identify special laundering requirements and appropriate hygienic cleaning options.¹¹⁸⁷

Although hot-water washing is an effective laundry disinfection method, the cost can be significant. Laundries are typically the largest users of hot water in hospitals, consuming 50% - 75% of the total hot water.¹¹⁸⁹ This represents an average of 10% - 15% of the energy used by a hospital. Several studies have shown that lower water temperatures of 22°C - 50°C (71°F - 77°F) can satisfactorily reduce microbial contamination when the cycling of the washer, the wash detergent, and the amount of bleach are carefully monitored and controlled.^{1161, 1190 - 1194} Low-temperature laundry cycles rely heavily on the presence of chlorine- or oxygen-activated bleach to reduce the levels of microbial contamination. The selection of hot- or cold-water laundry cycles may be dictated by state healthcare facility licensing standards or other regulation. Regardless of whether hot or cold water is used for washing, the temperatures reached in drying and especially during ironing provide additional significant microbiocidal action.¹¹⁶¹

After washing, cleaned and dried textiles, fabrics, and clothing are pressed, folded and packaged for transport, distribution, and storage by methods that ensure their cleanliness until use.² The transport of cleaned textiles and fabrics is handled separately from transport of contaminated laundry. State regulations and/or accrediting standards may dictate the procedures for this activity.

In the absence of microbiological standards for laundered linens, there is no rationale for routine microbiological sampling of cleaned healthcare textiles and fabrics.¹¹⁹⁵ Sampling may be used as part of an outbreak investigation if epidemiologic evidence suggests textiles, fabrics, or clothing as a vehicle for disease transmission. Sampling techniques include aseptically macerating the fabric into pieces and adding these to broth media, or using contact plates (RODAC plates) for direct surface sampling.^{1182, 1195} When evaluating the disinfecting properties of the laundering process specifically, placing pieces of fabric between two membrane filters may help to minimize the contribution of the physical removal of microorganisms.¹¹⁹⁶

Washing machines and dryers in residential care settings are more likely to be consumer items rather than the commercial, heavy-duty, large volume units typically found in hospitals and other institutional healthcare settings. Although all washing machines and dryers in healthcare settings must be properly maintained for performance according to the manufacturer's instructions, questions have been raised about the need to disinfect washers and dryers in residential care. Disinfection of the tubs and tumblers of these machines is unnecessary when proper laundry procedures are followed. These procedures involve the physical removal of bulk solids (e.g., feces) before the wash/dry cycle and proper use of temperature, detergent, and laundry additives. There have been no reports of infection linked to laundry procedures in residential care.

5. Special Laundry Situations

Some fabric items (e.g., surgical drapes, reusable gowns, and scrubs) need to be sterilized before use and therefore require steam autoclaving after laundering.⁷ Although the American Academy of Pediatrics in previous guidelines recommended autoclaving for linens in neonatal intensive care units (NICUs), studies on the microbial quality of

routinely cleaned NICU linen have not identified any increased risk of infection among the neonates receiving care.¹¹⁹⁷ Consequently, hygienically clean linens are suitable for use in this setting.⁹³⁹ The use of sterile linens in burn therapy units remains unresolved.

Items with rubberized backing are often used as personal protective equipment. When these items become contaminated with blood or other body substances, the manufacturer's instructions for decontamination and cleaning take into account the compatibility of the rubber backing with the chemical germicides or detergents used in the process. The directions for decontaminating these items should be followed as indicated; discard the item when the backing develops surface cracks and breaks.

Dry cleaning, a laundering process which utilizes organic solvents such as perchloroethylene for soil removal, is an alternative means of cleaning fabrics that might be damaged in conventional water and detergent washing. A number of studies, however, have shown that dry cleaning alone is relatively ineffective in reducing the numbers of bacteria and viruses on contaminated linens;^{1198, 1199} microbial populations are significantly reduced only when dry cleaned articles are heat pressed. Dry cleaning should therefore not be considered a routine option for healthcare facility laundry and should be reserved for those special circumstances for fabrics which cannot be safely cleaned with water and detergent.¹²⁰⁰

6. Surgical Gowns, Drapes and Disposable Fabrics

An issue of recent concern is the use of disposable (single use) versus reusable (multiple use) surgical attire and fabrics in health care.¹²⁰¹ Regardless of the material used to manufacture gowns and drapes, these items need to be impermeable to liquids and viruses.^{7, 1202, 1203} Repellency and pore size of the fabric contribute to gown performance,¹²⁰⁴ but repeated launderings of reusable gowns appeared to reduce the ability of the fabric to prevent transmission of bacteria.^{1205, 1206} Reinforced gowns (i.e., gowns with double-layered fabric) are generally more resistant to liquid strike-through.^{1207, 1208} In one study in Europe, surgeons overwhelmingly preferred reinforced disposable gowns to a reusable cotton gown based on the ability of the fabric to prevent liquid strike-through.¹²⁰⁸ Reinforced gowns may, however, be less comfortable. Guidelines for selection and use of barrier materials for surgical gowns and drapes have been published.¹²⁰⁹ Preferred product attributes include: 1) adequate barrier performance against liquids and microorganisms; 2) compatibility with reprocessing methods if reusable; 3) durability against tears and staining; 4) lack of toxicity; 5) low lint production; and 6) a positive cost-benefit ratio. It is the responsibility of the healthcare facility to assure optimal protection of patients and healthcare workers. Not all fabric items in healthcare lend themselves to single-use. Facilities exploring options for gowns and drapes should consider the expense of disposable items, and the impact on the facility's waste management costs once these items are discarded. Costs associated with the use of durable goods involve the fabric or textile items, the staff expenses to collect, sort, clean, and package the laundry, and the energy costs to operate the laundry if on-site or the costs to contract with an outside service.¹²¹⁰

7. Antimicrobial-Impregnated Articles

Manufacturers are increasingly incorporating antibacterial or antimicrobial chemicals into consumer and healthcare items. Some consumer products bearing labels that indicate treatment with antimicrobial chemicals have included pens, cutting boards, toys, household cleaners, hand lotions, cat litter, soaps, cotton swabs, toothbrushes, and cosmetics. The "antibacterial" label on household cleaning products, in particular, gives consumers the impression that the products perform "better" than comparable products without this labeling, when in fact all household cleaners have antibacterial properties.

In the healthcare setting, treated items include children's pajamas, mattresses, and bed linens with label claims of antimicrobial properties. These claims require careful evaluation to determine if they pertain to the use of antimicrobial chemicals as preservatives for the fabric or other components or if they imply a health claim.¹²¹¹ **At present there is no evidence that use of these products will make consumers and patients healthier or prevent disease.** There are no data to support the use of these items as part of a sound infection control strategy, and therefore there is little evidence to justify the additional expense expected if a facility were to replace its bedding and sheets with these treated products.

The EPA has reaffirmed its position that manufacturers who make public health claims for articles containing antimicrobial chemicals must provide evidence to support those claims as part of the registration process.¹²¹² Current

EPA regulations outlined in the Treated Articles Exemption of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) require manufacturers to register both the antimicrobial chemical used in or on the product and the finished product itself if a public health claim is maintained for the item. The exemption applies to the use of antimicrobial chemicals for the purpose of preserving the integrity of the product's raw material(s). The U.S. Federal Trade Commission (FTC) is evaluating manufacturer advertising of products with antimicrobial claims.¹²¹³

8. Standard Mattresses, Pillows, and Air-Fluidized Beds

Standard mattresses and pillows can become contaminated with body substances during patient care if the integrity of the covers of these items is compromised. The practice of sticking needles into the mattress should be avoided. Patches for tears and holes in mattress covers do not provide an impermeable surface over the mattress. Mattress covers should be replaced when torn; the mattress should be replaced if it is visibly stained. Wet mattresses, in particular, can be a significant environmental source of microorganisms. Infections and colonizations due to *Acinetobacter* spp., methicillin-resistant *Staphylococcus aureus* (MRSA), and *Pseudomonas aeruginosa* have been described, especially among burn patients.^{1214 - 1219} In these reports, the removal of wet mattresses was an important infection control measure, and efforts were made to ensure that pads and covers were cleaned and disinfected between patients, using disinfectant products compatible with mattress-cover materials so that these covers remained impermeable to fluids.^{1214 - 1218} Pillows and their covers should be easily cleanable, preferably in a hot water laundry cycle.¹²¹⁹

Air-fluidized beds are used for the care of patients immobilized for extended periods of time because of therapy or injury (e.g., pain, decubitus ulcers, burns).¹²²⁰ These specialized beds consist of a base unit filled with microsphere beads fluidized by warm, dry air flowing upward from a diffuser located at the bottom of the unit. A porous, polyester filter sheet separates the patient from direct contact with the beads but allows body fluids to pass through to the beads. Moist beads aggregate into clumps which settle to the bottom where they are removed as part of routine bed maintenance.

Because the beads become contaminated with the patient's body substances, concerns have been raised about the potential for these beds to serve as an environmental source of pathogens. Pathogens such as *Enterococcus* spp., *Serratia marcescens*, *Staphylococcus aureus*, and *Streptococcus fecalis* have been recovered either from the microsphere beads or the polyester sheet after cleaning.^{1221, 1222} Reports of cross-contamination of patients, however, are few.¹²²² Nevertheless, routine maintenance and between-patient decontamination procedures are important to minimize potential risks to patients. Regular removal of bead clumps, coupled with the warm, dry air of the bed can help to minimize bacterial growth in the unit.^{1223 - 1225} Beads are decontaminated in between patients by high heat (range 45°C - 90°C [113°F - 194°F], depending on the manufacturer's specifications) for at least 1 hour; this is especially important for the inactivation of *Enterococcus* spp. which are relatively resistant to heat.^{1226, 1227} The polyester filter sheet requires regular changing and thorough cleaning and disinfection, especially between patients.^{1221, 1222, 1226, 1227}

Microbial contamination of the air space in the immediate vicinity of a properly maintained air-fluidized bed is similar to that found in air around conventional bedding, even though air flows out of the base unit and around the patient.^{1224, 1228, 1229} An operational air-fluidized bed may, however, interfere with proper pressure differentials, especially in negative-pressure rooms.¹²³⁰ The effect varies with the location of the bed relative to the room's configuration and supply and exhaust vent locations. Use of an air-fluidized bed in a negative-pressure room requires consultation with a facility engineer to determine appropriate placement of the bed.

H. Animals in Healthcare Facilities

1. General Information

The increasing number of animals in healthcare facilities has prompted consideration for transmission of zoonotic pathogens from animals to humans in these settings. Animals in healthcare facilities have traditionally been limited to laboratories and research areas. Their presence in patient-care areas is now more frequent, both in acute-care and long-term care settings. Although dogs and cats are commonly encountered in healthcare settings, other animals (e.g., fish, birds, non-human primates, rabbits, rodents, reptiles) can also be present as research, resident, or service animals. These animals can serve as sources of zoonotic pathogens that could potentially infect patients and healthcare workers (Table 39).^{1231 - 1244} There is the potential for animals to serve as reservoirs for antibiotic-resistant microorganisms which could be introduced to the healthcare setting while the animal is present. Vancomycin-resistant enterococci (VRE) have been isolated from both farm animals and pets,¹²⁴⁵ and a cat in a geriatric care center was found to be colonized with methicillin-resistant *Staphylococcus aureus* (MRSA).¹²⁴⁶

Table 39. Examples of Diseases Associated with Zoonotic Transmission^a

Infectious Disease	Cats	Dogs	Fish	Birds	Rabbits	Reptiles ^b	Primates	Rodents ^b
Virus								
Lymphocytic choriomeningitis								[
Rabies	[[
Bacteria								
Campylobacteriosis	[[[[[
<i>C. canimorsus</i> infection	[[
Cat scratch disease (<i>Bartonella henselae</i>)	[
Leptospirosis	[[[
Mycobacteriosis			[[[
Pasteurellosis	[[[
Plague	[[[[
Psittacosis				[
Q fever (<i>Coxiella burnetii</i>)	[
Rat bite fever (<i>Spirillum minus</i> , <i>Streptobacillus moniliformis</i>)								[
Salmonellosis	[[[[[[[
Tularemia	[[[
Yersiniosis					[[[[
Parasites								
Ancylostomiasis	[[[
Cryptosporidiosis	[
Giardiasis	[[[
Toxocariasis	[[[
Toxoplasmosis	[[[
Fungi								
Blastomycosis		[
Dermatophytosis		[[[[

a. Adapted from Reference 1235 and used with permission of the publisher. This table does not include vectorborne diseases.

b. Reptiles include lizards, snakes, and turtles. Rodents include hamsters, mice, and rats.

[= pathogen isolated from animals and considered potential risk to humans.

Zoonoses can be transmitted from animal to man either directly or indirectly via bites, scratches, aerosols, ectoparasites, accidental ingestion, or contact with contaminated soil, food, water, or unpasteurized milk.^{1235, 1236, 1247 - 1249} Colonization of healthcare workers and hand transferral of pathogens acquired from pets in the home represent potential sources and modes of transmission of zoonotic pathogens in healthcare settings. Two examples of these situations include an outbreak among neonates of infections due to a yeast, *Malassezia pachydermatis* traced to transfer of the yeast from the hands of healthcare workers with pet dogs at home,¹²⁵⁰ and an outbreak of *Rhodococcus (Gordona) bronchialis* sternal SSIs after coronary-artery bypass surgery traced to a colonized nurse whose dogs were culture-positive for the

organism.¹²⁵¹ In the latter instance it was not determined whether the dogs were the sole source of the organism or if there were other environmental reservoirs. Nonetheless, there is minimal evidence that outbreaks of infectious diseases have occurred as a result of contact with animals in areas housing immunocompetent patients. This may, however, be due in part to relatively limited presence of the animals in healthcare facilities in the past (e.g., supervised visitation) and the fact that patients involved in those encounters were not immunosuppressed. Formal scientific studies to evaluate potential risks of transmission of zoonoses in healthcare settings outside of the laboratory are lacking.

2. Pet Visitation, Pet Therapy, and Resident Animals

Pet visitation programs allow patients and pets to visit in a central or common area location in the facility rather than have the animals come to individual patient rooms. A group session with the animals enhances opportunities for patients and facility residents to interact with caregivers, family members, and volunteers.^{1252 - 1254}

Pet therapy, or pet-assisted therapy, is a goal-directed intervention that incorporates an animal into the treatment process.^{1234, 1235} The concept for pet therapy arose from the observation that patients with pets at home appear to recover from surgical and medical procedures more rapidly compared to patients without pets.^{1255, 1256} The consensus in health care is that contact with pets is beneficial for enhancing wellness in certain patient populations, (e.g., children, the elderly, extended-care hospitalized patients).^{1252, 1257 - 1260} This consensus, however, is largely derived from anecdotal reports and observations of patient/animal interactions.^{1260 - 1262} Guidelines for establishing pet therapy programs are available for facilities considering this option.¹²⁶³

Animals participating in pet visitation or pet therapy sessions should have be current and complete with regard to recommended immunizations; they should be in good health. Animals should be routinely screened for enteric parasites and/or have evidence of a recently completed anthelmintic regimen.¹²⁶⁴ They should also be free of ectoparasites (e.g., fleas, ticks) and should have no obvious dermatologic lesions that could be associated with bacterial, fungal, or viral infections or parasitic infestations. Animals should be clean and well-groomed. The visits must be supervised by persons who know the animals and their behavior, and the area must be cleaned after visits according to standard cleaning procedures.

The most important infection control measure to prevent potential disease transmission is strict enforcement of handwashing or hand hygiene measures (using alcohol-based hand degerming agents when a sink is not available) for all patients, staff, and residents after handling the animals.¹²⁵⁸ Care should also be taken to avoid direct contact with animal urine or feces. Clean-up of these substances from environmental surfaces requires gloves and the use of leak-resistant (e.g., “zip-lockable”) plastic bags to discard absorbent material used in the process.²

The American Academy of Allergy, Asthma, and Immunology estimates that dog or cat allergies occur in approximately 15% of the population.¹²⁶⁵ Minimizing contact with animal saliva, dander, and/or urine helps to mitigate allergic responses.^{1265 - 1267} Some facilities may not allow animal visitation for patients with: 1) underlying asthma; 2) recognized allergies to cat or dog hair; 3) respiratory allergies of unknown etiology; and 4) immunosuppressive disorders. Hair shedding can be minimized by processes that remove dead hair (e.g., grooming) and that prevent the shedding of dead hair (e.g., therapy capes for dogs). Allergens can be minimized by bathing therapy animals within two days of a visit.¹²⁶⁸

Animal programs require precautions to prevent bites. Common pathogens associated with animal bites include *Capnocytophaga canimorsus*, *Pasteurella* spp., *Staphylococcus* spp., and *Streptococcus* spp. Selecting well-behaved dogs for these programs will greatly decrease the incidence of bites. Rodents, exotic species, and wild animals must be handled with caution. If a bite does occur, the wound must be cleansed immediately and monitored for subsequent infection. Most infections can be treated with antibiotics, and often antibiotics are prescribed presumptively or prophylactically in these situations.

Immunocompromised patients may be at higher risk of acquiring some pet-related zoonoses. Although guidelines have been developed to minimize the risk of disease transmission to HIV-infected patients,⁸ these recommendations may be applicable for patients with other immunosuppressive disorders. In addition to handwashing or hand hygiene, these recommendations include avoiding contact with: 1) animal feces and soiled litter box materials; 2) animals with diarrhea; 3) very young animals [<6 months of age, <1 year old for cats]; and 4) exotic animals and reptiles.⁸ Pets with diarrhea

should receive veterinary care to resolve their condition. Many facilities do not offer pet visitation or pet therapy programs for severely neutropenic patients (e.g., HSCT patients, patients on corticosteroid therapy) or exclude these patients from participating in pet visitation programs.¹²⁵² The question of whether family pets can visit terminally-ill HSCT patients or other severely immunosuppressed patients is best handled on a case-by-case basis, although animals should not be brought into the HSCT unit or any other unit housing severely immunosuppressed patients.

Many healthcare facilities are adopting more home-like environments for residential-care or extended-stay patients in acute-care settings, and resident animals are one element of this approach.¹²⁶⁹ One concept, the “Eden Alternative,” incorporates children, plants, and animals (e.g., dogs, cats, fish, birds, rabbits, rodents) into the daily care setting.^{1270, 1271} The use of resident animals has not been scientifically evaluated, and several issues beyond the benefits of therapy need to be considered before embarking on such a program. These include: 1) whether animals will come into direct contact with patients; 2) whether animals will be allowed to roam freely in the facility; 3) how staff will provide care for the animals; 4) how staff will deal with allergies, asthma, and phobias; and 5) how staff will prevent bites and scratches; and 6) how staff will prevent soil and environmental by enteric parasites [e.g., *Toxoplasma*, *Toxocara*, and *Ancylostoma*].^{1272, 1273} As a general preventive measure, resident animal programs are advised to restrict animals from: 1) food preparation kitchens; 2) laundries; 3) central sterile supply and any storage areas for clean supplies; 4) medication preparation areas; 5) isolation and protective environments; 6) operating rooms; and 7) patient eating areas. Handwashing or use of alcohol hand gels by patients and staff should be routine after contact with animals.

3. Service Animals

A service animal is any animal individually trained to do work or perform tasks for the benefit of a person with a disability.^{1266, 1274} A service animal is not considered a pet but rather an animal trained to help its handler overcome limitations of his/her disability. Title III of the “Americans with Disabilities Act” of 1990 mandates that persons with disabilities accompanied by service animals generally be allowed access with their service animals into places of public accommodation, including restaurants, public transportation, schools, and healthcare facilities.^{1266, 1274}

An overview of the subject of service animals and their presence in healthcare facilities has been published;¹²⁶⁶ Since a healthcare facility is considered a public place, a service animal may accompany its handler within the facility unless the animal’s presence or behavior creates a fundamental alteration or a direct threat to other persons or to the nature of the goods and services provided.¹²⁶⁶ Requiring documentation for access of a service animal to an area generally accessible to the public could potentially be viewed as an unfair burden on a disabled person. If persons are permitted to enter care areas without additional precautions to prevent transmission of infectious agents (e.g., donning gloves, gowns, or masks), a clean, healthy, well-behaved service animal should be allowed access with its handler.¹²⁶⁶ Similarly, if immunocompromised patients are able to receive visitors without using protective garments or equipment, then there is little justification to deny access to service animals and their handlers to the area.¹²⁶⁶ Care areas where service animal access might be restricted include: 1) isolation areas; 2) care areas for immunosuppressed patients; 3) intensive care units; 4) operating rooms; and 5) burn therapy units.¹²³⁵ If a service animal must be separated from its handler during the provision of outpatient health care, it is important that the animal be supervised by a responsible person, or if this is not possible, to place the animal in a crate or carrier.¹²⁶⁶ If the animal’s handler is admitted and would not be expected to engage in activities that would involve the service animal, then it would be reasonable for the service animal to be returned to the handler’s residence until the patient is discharged.

Although animals may potentially carry zoonotic pathogens transmissible to man, the risk is minimal with a healthy, clean, vaccinated, well-behaved, and well-trained service animal, the most common of which are dogs and cats. There have been no published reports of human infectious disease originating in service dogs. The animal health recommendations and infection control measures used during service animal encounters are identical to those pet visitations and pet therapy. Measures used to minimize the impact of allergies among patients and staff to the service animal are similar to those used in pet therapy situations. No special clean-up procedures are needed to clean the area visited by a service animal; cleaning methods and procedures would be the same as those used to clean after pet therapy.

The use of exotic animals (e.g., reptiles, non-human primates) as service animals is problematic. Concerns about these animals are discussed in two excellent reviews.^{1235, 1266} Because of high-risk behaviors and potential for zoonotic disease transmission (e.g., herpes B infection), nonhuman primates are not recommended as service animals, especially if the

primates are in contact with the general public. Healthcare administrators should consult the Americans with Disabilities Act for guidance when developing policies about service animals in their facilities.^{1266, 1274}

4. Animals as Patients in Human Healthcare Facilities

The potential for direct and indirect transmission of zoonoses must be considered when rooms and equipment in human healthcare facilities are used for the medical or surgical treatment or diagnosis of animals. Inquiries should be made to veterinary hospitals to determine an appropriate facility and equipment to care for an animal. If human healthcare facilities must be used for animal treatment or diagnostics, these guidelines are suggested: 1) avoid the use of operating rooms or other rooms used for invasive procedures [e.g., cardiac catheterization labs, invasive nuclear medicine areas]; 2) use only disposable equipment or equipment that can be cleaned and disinfected or sterilized; and 3) once medical or surgical instruments are used on animals, reserve these instruments for future use only on animals.

5. Research Animals in Healthcare Facilities

The risk of acquiring a zoonotic infection from research animals has decreased in recent years because many small laboratory animals (e.g., mice, rats, rabbits) now come from quality stock, many with defined microbiologic profiles.¹²⁷⁵ Larger animals (e.g., nonhuman primates) are still obtained frequently from the wild and may harbor pathogens transmissible to humans. Primates, in particular, benefit from vaccinations to protect their health during the research period, provided the vaccination doesn't interfere with study of the particular agent. Animals serving as models for human disease studies pose some risk for transmission of infection to laboratory or healthcare workers from percutaneous or mucosal exposure. Exposures can occur either through direct contact with an infected animal or its body substances and secretions, or indirect contact with infectious material on equipment, instruments, surfaces, or supplies.¹²⁷⁵ Uncontained aerosols generated during laboratory procedures can also transmit infection.

Infection control measures to prevent transmission of zoonotic infections from research animals are largely derived from basic lab safety principles. These include: 1) purchasing pathogen-free animals; 2) quarantining incoming animals to detect any zoonotic pathogens; 3) treating infected animals or removing them from the facility; 4) vaccinating animal carriers and high-risk contacts if possible; 5) using specialized containment caging or facilities; and 6) using protective clothing and equipment [e.g., gloves, face shields, gowns, masks].¹²⁷⁵ An excellent resource for detailed discussion of these safety measures has been published.⁹⁵²

The animal research unit within a healthcare facility should be engineered to provide: 1) adequate containment of animals and pathogens; 2) daily decontamination and transport of equipment and waste; 3) proper ventilation and air filtration which prevents recirculation of the air in the unit to other areas of the facility; and 4) negative air pressure in the animal rooms relative to the corridors. To ensure adequate security and containment, there should be no through traffic to other areas of the healthcare facility; access should be restricted to animal care staff, researchers, environmental services, maintenance, and security.

Occupational health programs for animal care staff, researchers, and maintenance staff should take into consideration the animals' natural pathogens and research pathogens. Components of such programs include but are not limited to: 1) prophylactic vaccines; 2) TB skin testing when primates are used; 3) baseline serums; and 4) hearing and respiratory testing. Work practices, personal protective equipment, and engineering controls specific for each of the four animal biosafety levels have been published.⁹⁵² The facility's occupational or employee health clinic should be aware of the appropriate post-exposure procedures involving zoonoses and have the appropriate post-exposure biologicals and medications on hand.

Animal research area staff should also develop standard operating procedures for: 1) daily animal husbandry [protection of the employee while facilitating animal welfare]; 2) pathogen containment and decontamination; 3) management, cleaning, disinfecting and/or sterilizing equipment and instruments; and 4) employee training for laboratory safety and safety procedures specific to animal research worksites.⁹⁵² The federal Animal Welfare Act of 1966 and its amendments serves as the regulatory basis for ensuring animal welfare in research.^{1276, 1277}

I. Regulated Medical Waste

1. Epidemiology

There is no epidemiologic evidence to suggest that waste from hospitals, other healthcare facilities, or clinical/research laboratories is any more infective than residential waste. Several studies have compared the microbial load and the diversity of microorganisms in residential wastes and wastes from a variety of healthcare settings.^{1278 - 1284} Although hospital wastes had a greater number of different bacterial species compared to residential waste, wastes from residences were more heavily contaminated.^{1279, 1280} Moreover, there is no epidemiologic evidence that traditional waste-disposal practices of healthcare facilities (i.e., for which clinical and microbiological wastes were decontaminated on site before leaving the facility) have caused disease in either the healthcare setting or the general community.^{1282, 1283} This statement excludes, however, sharps injuries sustained during or immediately after the delivery of patient care before the sharp is “discarded.” Therefore, identifying wastes for which handling and disposal precautions are indicated is largely a matter of judgment about the relative risk of disease transmission, since there are no reasonable standards on which to base these determinations. Aesthetic and emotional considerations, originating during the early years of the HIV epidemic, have, however, figured into the development of treatment and disposal policies, particularly for pathology and anatomy wastes and sharps.^{1284 - 1287} Public concerns have resulted in the promulgation of federal, state, and local rules and regulations regarding medical waste management and disposal.^{1288 - 1293}

2. Categories of Medical Waste

A precise definition of medical waste based on the quantity and type of etiologic agents present is virtually impossible. The most practical approach to medical waste management is to identify wastes that represent a sufficient potential risk of causing infection during handling and disposal and for which some precautions appear prudent. Healthcare facility medical wastes targeted for handling and disposal precautions include microbiology laboratory waste (e.g., microbiologic cultures and stocks of microorganisms), pathology and anatomy waste, blood, blood specimens from clinics and laboratories, blood products and other body fluid specimens.² Moreover, the risk of either injury or infection from certain sharp items (e.g., needles, scalpel blades) contaminated with blood also needs to be considered. Although any item that has had contact with blood, exudates, or secretions may be potentially infective, it is not normally considered practical or necessary to treat all such waste as infective. Federal, state, and local guidelines and regulations specify the categories of medical waste that are subject to regulation and outline the requirements associated with treatment and disposal. The categorization of these wastes has generated the term “regulated medical waste” to draw attention to the role of regulation in defining the actual material and as an alternative to “infectious waste,” given the lack of evidence of infectivity. The EPA’s *Manual for Infectious Waste Management* identifies and categorizes other specific types of waste generated in healthcare facilities with research laboratories which also require handling precautions.¹²⁸⁸

3. Management of Regulated Medical Waste in Healthcare Facilities

Medical wastes require careful disposal and containment before collection and consolidation for treatment. OSHA has dictated initial measures for discarding regulated medical waste items. These measures are designed to protect the workers who generate medical wastes and who manage the wastes from point of generation to disposal.⁹¹¹ A single leak-resistant biohazard bag is usually adequate for containment of regulated medical wastes, provided the bag is sturdy and the waste can be discarded without contaminating the bag’s exterior. Contaminating or puncturing of the bag requires placement into a second biohazard bag. All bags should be securely closed for disposal. Puncture-resistant containers located at the point of use (e.g., sharps containers) are used as containment for discarded slides or tubes with small amounts of blood, scalpel blades, needles and syringes, and unused sterile sharps.⁹¹¹ To prevent needlestick injuries, needles should not be recapped, purposefully bent, or broken by hand. CDC (including NIOSH) has published general guidelines for handling sharps.^{6, 1294} Healthcare facilities may need additional precautions to prevent the production of aerosols during handling blood-contaminated items for certain rare diseases or conditions such as Lassa fever or Ebola virus infection.²⁰⁰

It is often necessary to transport or store regulated medical wastes within the healthcare facility prior to terminal treatment. EPA guidelines, in addition to state regulations, address the safe transport and storage of regulated medical wastes, both on-site and off-site.¹²⁸⁸ Healthcare facilities are instructed to dispose medical wastes regularly to avoid accumulation. Medical wastes requiring storage should be kept in labeled, leak-proof, puncture-resistant containers under conditions that minimize or prevent obnoxious odors. The storage area should be well ventilated and be inaccessible to vertebrate pests. Any facility that generates regulated medical wastes should have a regulated medical waste management plan to assure health and environmental safety as per federal, state, and local regulations.

4. Treatment of Regulated Medical Waste

Regulated medical wastes are treated or decontaminated to reduce the microbial load in or on the waste and to render the by-products safe for further handling and disposal. From a microbiological standpoint, there is no need to render the waste “sterile” since the treated waste will not be deposited in a sterile site, nor is there the need to subject waste to the same reprocessing standards as are determined for surgical instruments. Historically, treatment methods involved steam-sterilization (autoclaving), incineration, or interment (for anatomy wastes). Alternative treatment methods developed in recent years include, but are not limited to chemical disinfection, grinding/shredding/disinfection methods, energy-based technologies (e.g., microwave or radiowave treatments), and disinfection/encapsulation methods.¹²⁸⁹ State medical waste regulations specify appropriate treatment methods for each category of regulated medical waste.

Of all the categories comprising regulated medical waste, microbiological wastes (e.g., untreated cultures, stocks, amplified microbial populations) pose the greatest potential for infectious disease transmission, and sharps pose the greatest risk for injuries. Untreated stocks and cultures of microorganisms are subsets of the clinical laboratory or microbiological waste stream. If the microorganism must be grown and amplified in culture to high concentration in order to work with the specimen, then this is an item that should be considered for on-site decontamination, preferably within the laboratory unit, by containing the waste and autoclaving. If steam sterilization in the healthcare facility is used for treatment, exposure of the waste for up to 90 minutes at 121°C (250°F) in an autoclave, depending on the size of the load and type container, may be necessary to assure an adequate decontamination cycle.^{1295 - 1297} After steam sterilization, the residue can be safely handled and discarded with all other nonhazardous solid waste. On-site incineration is another treatment option for microbiological waste, pathological and anatomical waste, provided the incinerator is engineered to completely burn these wastes and stay within EPA emissions standards.¹²⁹⁰ Improper incineration of waste with high moisture and low energy content (e.g., pathology waste), can lead to emission problems.

Concerns have been raised about the ability of modern healthcare facilities to inactivate microbiological wastes on-site, given that many of these institutions have decommissioned their laboratory autoclaves. Current laboratory guidelines for working with infectious microorganisms at biosafety level (BSL) 3 recommend that all laboratory waste be decontaminated before disposal by an approved method and that it is preferred that this decontamination be done within the laboratory.⁹⁵² These same guidelines recommend that all materials removed from a BSL 4 laboratory (unless they are biological materials that are to remain viable) are to be decontaminated before they leave the laboratory.⁹⁵² Recent federal regulations for laboratories that work with certain biological agents known as “select agents” (i.e., those that have the potential to pose a severe threat to public health and safety) are required to be destroyed on-site before disposal as well as isolates of select agents that are from a clinical specimen intended for diagnostic, reference, or verification purposes.¹²⁹² Though recommendations for laboratory waste disposal from BSL 1 or 2 laboratories (most healthcare clinical and diagnostic laboratories are designated as BSL 2) allow for these materials to be decontaminated off-site before disposal, on-site decontamination by a known effective method would be preferred to reduce the potential of exposure during the handling of infectious material.

A recent outbreak of TB among workers in a regional medical waste treatment facility in the United States demonstrated the hazards associated with aerosolized microbiological wastes.^{1298, 1299} The facility received untreated diagnostic cultures of *Mycobacterium tuberculosis* from several different healthcare facilities. The regional facility treated waste with a grinding/shredding process that produced aerosols of the material as it was introduced into the machine before chemical disinfection.^{1298, 1299} Several operational deficiencies facilitated the release of aerosols and exposed workers to airborne *M. tuberculosis*. Among the suggested control measures was that healthcare facilities perform on-site decontamination of laboratory waste containing live cultures of microorganisms before release of the waste to a waste management company.^{1298, 1299} This is supported by the recommendations found in the CDC/NIH guideline for laboratory workers.⁹⁵² This outbreak demonstrates the need to avoid the use of any medical waste treatment method or technology that can aerosolize pathogens from live cultures and stocks (especially those of airborne microorganisms) unless aerosols can be effectively contained and workers can be equipped with proper personal protective equipment.¹²⁹⁸ - ¹³⁰⁰ Safe laboratory practices, including waste management, have been published.^{952, 1301}

In an era when local, state, and federal healthcare facilities and laboratories are developing bioterrorism response strategies and capabilities, the need to reinstate in-laboratory capacity to destroy cultures and stocks of microorganisms becomes an important issue. Recent federal regulations require healthcare facility laboratories to maintain the capability

of destroying discarded cultures and stocks on-site if these laboratories isolate any of the microorganisms or toxins identified as a “select agent” from a clinical specimen (Table 40).¹²⁹² As an alternative, isolated cultures of select agents can be transferred to a facility registered to accept these agents

Table 40. Microorganisms and Biologicals Identified as Select Agents¹²⁹²

Viruses	Crimean-Congo hemorrhagic fever virus; Eastern equine encephalitis virus; Ebola viruses; Equine morbillivirus; Lassa fever virus; Marburg virus; Rift Valley fever virus; South American hemorrhagic fever viruses (Junin, Machupo, Sabia, Flexal, Guanarito); Tick-borne encephalitis complex viruses; Variola major (smallpox) virus; Venezuelan equine encephalitis virus; viruses causing hantavirus pulmonary syndrome; Yellow fever virus
Exemptions	Vaccine strains of viral agents (Junin virus strain candid.#1; Rift Valley fever virus strain MP-12; Venezuelan equine encephalitis virus strain TC-83; Yellow fever virus strain 17-D)
Bacteria	<i>Bacillus anthracis</i> ; <i>Brucella abortus</i> , <i>B. melitensis</i> , <i>B. suis</i> ; <i>Burkholderia (Pseudomonas) mallei</i> , <i>B. (Pseudomonas) pseudomallei</i> ; <i>Clostridium botulinum</i> ; <i>Francisella tularensis</i> ; <i>Yersinia pestis</i>
Exemptions	Vaccine strains as described in Title 9 CFR 78.1
Rickettsiae	<i>Coxiella burnetii</i> ; <i>Rickettsia prowazekii</i> , <i>R. rickettsii</i>
Fungi	<i>Coccidioides immitis</i>
Toxins	Abrin; Aflatoxins; Botulinum toxins; <i>Clostridium perfringens</i> epsilon toxin; Conotoxins; Diacetoxyscirpenol; Ricin; Saxitoxin; Shigatoxin; Staphylococcal enterotoxins; Tetrodotxin; T-2 toxin
Exemptions	Toxins for medical uses, inactivated for use as vaccines, or toxin preparations for biomedical research use at an LD ₅₀ for vertebrates of more than 100 nanograms per kilogram body weight are exempt. National standard toxins required for biologic potency testing as described in Title 9 CFR 113 are exempt.

in accordance with federal regulations.¹²⁹² State medical waste regulations can, however, complicate or completely prevent this transfer if these cultures are determined to be medical waste, as most states regulate the interfacility transfer of untreated medical wastes.

5. Discharging Blood, Fluids to Sanitary Sewers or Septic Tanks

All containers with more than a few milliliters of blood remaining after laboratory procedures, suction fluids, or bulk blood may be inactivated in accordance with state-approved treatment technologies, or the contents can be carefully poured down a utility sink drain or toilet.¹²⁹³ State regulations may dictate the maximum volume allowable for discharge of blood/body fluids to the sanitary sewer. There is no evidence that bloodborne diseases have been transmitted from contact with raw or treated sewage. Many bloodborne pathogens, particularly bloodborne viruses, are not stable in the environment for long periods of time,^{1302, 1303} and the discharge of small quantities of blood and other body fluids to the sanitary sewer is considered a safe method of disposing of these waste materials.¹²⁹³ Several factors enhance the likelihood of that bloodborne pathogens will be inactivated in the disposal process: 1) dilution of the discharged materials with water; 2) inactivation of pathogens due to exposure to cleaning chemicals, disinfectants, and other chemicals in raw sewage; and 3) effectiveness of sewage treatment in inactivating any residual bloodborne pathogens that reach the treatment facility. Small amounts of blood and other body fluids should not pose undue hardships on a municipal sewer system. Large quantities of these fluids, with their high protein content, might interfere with the biological oxygen demand (BOD) of the system. Local municipal sewage treatment restrictions may dictate that an alternative method of bulk fluid disposal be selected. State regulations may dictate what quantity constitutes a small amount of blood or body fluids.

Although concerns have been raised about the discharge of blood and other body fluids to a septic tank system, there is no evidence that septic tanks have served to transmit bloodborne infections. A properly functioning septic system will be adequate for inactivating bloodborne pathogens. System manufacturers’ instructions specify what materials may be discharged to the septic tank without jeopardizing its proper operation.

6. Medical Waste and CJD

Concerns have also been raised about the need for special handling and treatment procedures for wastes generated during the care of patients with CJD or other transmissible spongiform encephalopathies (TSEs). These concerns stem from the fact that the prion agents which cause TSEs appear to have significant resistance to inactivation by a variety of physical, chemical, or gaseous methods.¹³⁰⁴ There is no epidemiologic evidence, however, linking acquisition of CJD with medical waste disposal practices. Although it is prudent to handle neurologic tissue for pathologic examination and autopsy materials with care, using barrier precautions and specific procedures for the autopsy,¹¹¹³ there is no justification for using extraordinary measures once the materials are discarded. Regulated medical wastes generated during the care of the CJD patient can be managed using the same strategies as for wastes generated during the care of other patients. These wastes may be then disposed of in the sanitary landfill after decontamination or discharged to the sanitary sewer as appropriate.

II. Recommendations for Environmental Infection Control in Healthcare Facilities

A. Rationale

As in previous CDC guidelines, each recommendation is categorized on the basis of existing scientific data, theoretical rationale, applicability, and possible economic impact. The HICPAC system for categorizing recommendations has been modified to include a designation for engineering standards and actions required by state or federal regulations. Some of the recommendation statements of this guideline are largely derived from experience gained from situations that cannot be easily studied (e.g., floods). Guidelines and standards published by the American Institute of Architects (AIA) and the American Society of Heating, Refrigerating, and Air-conditioning Engineers (ASHRAE) form the basis of many of the recommendations. These publications include the AIA *Guidelines for Design and Construction of Hospitals and Health Care Facilities*¹²⁰ and the ASHRAE guidelines entitled *Ventilation for Acceptable Indoor Air Quality and Minimizing the Risk of Legionellosis Associated with Building Water Systems*.^{210, 637} Standards for a variety of engineered systems (e.g., air handling systems, cooling towers) are promulgated by ASHRAE based on engineer member input and CDC consultation.

B. Rankings

Recommendations are categorized according to the following designations:

Category IA - Strongly recommended for implementation and strongly supported by well-designed experimental, clinical, or epidemiological studies.

Category IB - Strongly recommended for implementation and supported by some experimental, clinical, or epidemiological studies and a strong theoretical rationale.

Category IC - Required by state or federal regulations, rules, or standards.

(The acronym of the promulgating federal agency is listed in parentheses when the statement is derived from a regulation. When a statement refers to regulation at the state level, the word "states" appears in parentheses.)

Category II - Suggested for implementation and supported by suggestive clinical or epidemiological studies or a theoretical rationale.

No recommendation - Unresolved issue. Practices for which insufficient evidence or no consensus regarding efficacy exists.

C. Recommendations

I. Air

1-1 Air-Handling Systems in Healthcare Facilities

- 1-1.1 In the absence of locally recommended standards, use ANSI/ASHRAE Standard 62, *Ventilation for Acceptable Indoor Air Quality*, as a ventilation standard for healthcare facilities.^{210, 234} *Category IC*
- 1-1.2 Monitor ventilation systems in accordance with engineering and manufacturers' recommendations to ensure process management through preventive engineering, optimal performance for removal of particulates, and elimination of excess moisture.^{35, 214} *Category IC*
- 1-1.2.a Ensure that HVAC filters are properly installed and maintained to prevent air leakages and dust overloads.^{17, 18, 106, 215} *Category IC*
- 1-1.2.b Monitor areas with special ventilation requirements (e.g., AII, PE) for ACH, and pressure differentials; document pressure differentials.^{21, 120, 241, 242, 263 - 265, 267} *Category IB*
- 1-1.2.c Engineer humidity controls into the HVAC system and monitor these to ensure proper moisture removal.¹²⁰ *Category IC*
- i Locate duct humidifiers upstream of the final filters. *Category IC*
- ii Incorporate a water-removal mechanism into the system. *Category IC*
- iii Locate all duct takeoffs sufficiently downstream of the humidifier so that moisture is completely absorbed. *Category IC*
- iv Incorporate steam humidifiers if possible. *Category II*
- 1-1.2.d Ensure that air intakes and exhaust outlets are located correctly in construction of new facilities and renovation of existing facilities.^{3, 120} *Category IC*
- i Locate exhaust outlets >25 feet from air-intake systems. *Category IC*
- ii Locate outdoor air intakes at least 6 feet above ground or 3 feet above roof level. *Category IC*
- iii Locate exhaust outlets from contaminated areas above roof level to minimize recirculation of exhausted air. *Category IC*
- 1-1.2.e Maintain the air intakes and inspect filters periodically to ensure proper operation.^{3, 120, 241, 242, 263 -265, 267} *Category IC*
- 1-1.2.f Remove bird roosts and nests near air intakes and prevent birds from gaining access to these structures.^{3, 98, 102, 104, 119} *Category IB*
- 1-1.2.g Prevent dust accumulation by regularly cleaning air-duct grilles when rooms are not occupied by patients.^{21, 120, 241, 242, 263 - 265, 267} *Category IC*
- 1-1.3 Use portable, industrial-grade HEPA filters (e.g., 300-800 ft³/min.) to augment the removal of respirable particles as needed.²¹³ *Category II*
- 1-1.3.a Select portable HEPA filters that can recirculate all or nearly all of the room air and provide ≥ 12 ACH.⁴ *Category IC*
- 1-1.3.b Do not use a portable HEPA filter unit in patient-care areas that was previously used in construction zones. *Category IC*
- 1-1.3.c Situate portable HEPA units so that all room air is filtered.⁴ *Category IC*
- 1-1.3.d Ensure that fresh-air requirements for the area are met.^{210, 213} *Category IC*
- 1-1.4 Follow proper procedures for use of rooms/areas with induction ventilation units.¹²⁰ *Category IC*
- 1-1.4.a Do not use these rooms/areas as PE.¹²⁰ *Category IC*
- 1-1.4.b Do not use a room with an induction ventilation unit as an AII room unless it can be demonstrated that all engineering controls required for AII are met.⁴ *Category IC*
- 1-1.5 Conduct a risk assessment and provide an adequate number of AII and PE rooms or areas to meet the needs of the patient population.^{4, 9, 120} *Category IC*
- 1-1.6 When using UVGI as a supplemental engineering control, install fixtures: 1) on the wall near the ceiling or suspended from the ceiling as an upper air unit; 2) in the air-return duct of an AII area; or 3) in designated enclosed areas or booths for sputum induction.⁴ *Category II*
- 1-1.7 Seal windows in buildings with centralized HVAC systems and especially in PE areas.^{35, 111} *Category IC*
- 1-1.8 Keep emergency doors and exits in PE closed except during an emergency; equip emergency doors and exits with alarms. *Category IC*
- 1-1.9 Do not shut down HVAC systems other than for maintenance, repair, testing of emergency back-up capacity, or new construction.^{120, 202} *Category IC*

- 1-1.9.a If the HVAC system must be shut down, do not inactivate the entire system at one time. *Category IC*
- 1-1.9.b Provide backup emergency power and redundant air-handling and pressurization systems to maintain filtration, constant ACH and pressure differentials in PE units, AII, operating rooms, and other critical care areas.^{9, 268} *Category IC*
- 1-1.9.c Bag dust-filled filters immediately upon removal to prevent dispersion of dust and fungal spores during transport to a disposal site.¹⁰⁶ *Category IB*
- 1-1.9.d Coordinate HVAC system maintenance with infection control staff to allow for relocation of immunocompromised patients if necessary. *Category II*
- 1-1.9.e Allow sufficient time for adequate ACH to clean the air once the system is operational. *Category IC*
- 1-1.9.f Develop a contingency plan for a general power failure, with emphasis on restoration of proper air-quality and ventilation conditions in AII, PE, operating rooms, emergency departments, and intensive-care units. *Category IC*
- 1-1.9.g Coordinate system start-ups with infection control staff to protect patients in PE units from bursts of mold spores.^{9, 35, 268} *Category IC*
- 1-1.10 Clean ventilation ducts as per the manufacturer's recommendations for maintaining HVAC performance. *Category IC*

1-2 Construction, Renovation, Remediation, Repair, and Demolition

- 1-2.1 Establish a multi-disciplinary team that includes infection control staff to coordinate demolition, construction, and renovation projects so that proactive preventive measures are considered at the inception of such projects.^{120, 241, 242, 263 - 267} *Category IC*
- 1-2.2 When planning hospital construction, renovation, repairs, maintenance, or demolition activities, conduct a risk assessment to determine if immunocompromised patients may be exposed to high ambient-air fungal spore counts from construction and renovation, and develop a contingency plan to prevent such exposures.^{20, 109, 120, 263 - 267} *Category IB*
- 1-2.3 Educate both the construction team and the healthcare staff in immunocompromised patient-care areas about the airborne infection risk associated with construction projects, dispersal of fungal spores during such activities, and methods to control the dissemination of fungal spores.^{3, 120, 241, 242, 263 - 267, 1305 - 1308} *Category II*
- 1-2.4 Incorporate mandatory adherence agreements for infection control into construction contracts, with penalties for non-compliance and mechanisms to ensure timely correction of problems.^{3, 120, 241, 263 - 267} *Category II*
- 1-2.5 Establish and maintain airborne disease surveillance during construction, renovation, repair, and demolition activities.³ *Category IB*
 - 1-2.5.a Using active surveillance, monitor for airborne infections in immunocompromised patients.^{3, 9} *Category IB*
 - 1-2.5.b Periodically review the facility's microbiologic, histopathologic, and postmortem data to identify additional cases. *Category IB*
 - 1-2.5.c If cases of aspergillosis or other healthcare-associated airborne fungal infections occur, aggressively pursue the diagnosis with tissue biopsies and cultures as feasible.^{3, 64, 65, 79, 120, 241, 263 - 267} *Category IB*
- 1-2.6 Conduct parametric monitoring of the ventilation system components to ensure proper performance (e.g., filtration efficiency, pressure differentials). *Category IC*
- 1-2.7 Implement infection control measures during construction and repair.^{96, 97, 266, 267} *Category IB*
 - 1-2.7.a Before construction, perform an infection control risk assessment to define the scope of the project and the need for barrier measures.^{120, 241, 263 - 267} *Category IB*
 - 1-2.7.b Ensure proper operation of the air-handling system in the affected area before construction and after erection of barriers but before the room or area is set to negative pressure.^{266, 268} *Category IB*
 - 1-2.7.c Implement infection control measures for external demolition and construction activities.^{120, 241, 263 - 267} *Category IB*
 - i Seal off adjacent air intakes, or if this is not possible or practical, check the low-efficiency (roughing) filter banks frequently to avoid build-up of particulates, and determine if the facility can operate temporarily on recirculated air. *Category IC*
 - ii Seal the windows, especially in PE areas. *Category IC*
 - iii Implement dust control measures: 1) mist dusty surfaces; 2) divert pedestrian traffic away from work zones; and 3) use tacky mats in the construction zone at the entrances. *Category IC*

- iv Relocate immunocompromised patients whose rooms are adjacent to work zones.
Category IB
- 1-2.7.d Avoid damaging the underground water system to prevent soil and dust contamination which, when aerosolized, may pose a risk of legionellosis and infections due to other environmental pathogens to immunocompromised patients.²⁹⁴ *Category IB*
- 1-2.7.e Implement infection control measures for internal construction activities.^{120, 241, 263 - 267} *Category IB*
 - i Construct barriers between patient-care and construction areas to prevent dust from entering patient-care areas; ensure that barriers are impermeable to fungal spores and in compliance with local fire codes.^{20, 49, 97, 1308} *Category IB*
 - ii Seal off and block return air vents if rigid barriers are used for containment.^{266, 267}
Category IB
- 1-2.7.f Perform these engineering and work-site related infection control measures as needed for internal construction, repairs, and renovations: *Category IB*
 - i Create and maintain negative air pressure in work zones relative to adjacent patient-care areas but ensure that required engineering controls for those adjacent areas are maintained (e.g., AII, PE).^{20, 49, 97, 109, 299} *Category IB*
 - ii Monitor negative airflow inside rigid barriers with a door manometer if possible, or perform smoke-tube testing; use an alarm device as an alternative indicator of faulty pressure differential here. *Category IB*
 - iii Use airborne particle sampling to establish a rank order assessment of the air-handling system during construction and to ensure the integrity of the construction barriers.^{35, 100}
Category IB
 - iv Seal windows in work zones if practical; use window chutes for disposal of large pieces of debris as needed, but ensure that the negative pressure differential for the area is maintained.¹²⁰ *Category IC*
 - v Direct pedestrian traffic from construction zones away from patient-care areas to minimize the dispersion of dust.^{20, 49, 97, 109, 111, 263 - 267} *Category IC*
 - vi Provide construction crews with: 1) designated entrances, corridors, and elevators; 2) essential services [e.g., toilet facilities, vending machines]; 3) protective clothing [e.g., coveralls, footgear, headgear] for travel to patient-care areas; and 4) a space or anteroom for changing clothing and storing equipment.^{120, 241, 263 - 267} *Category IC*
 - vii Clean work zones and their entrances daily: 1) wet-wipe tools and tool carts before their removal from the work zone; 2) place tacky mats inside the entrance; and 3) cover debris before removal from the work zone.^{120, 241, 263 - 267} *Category IC*
 - viii For minor jobs in patient-care areas that include removal of ceiling tiles and disruption of the space above the false ceiling, use plastic sheets or prefabricated plastic units to contain dust; use a negative pressure system within this enclosure to remove dust and pass air through an industrial grade, portable HEPA filter (e.g., 300-800 ft³/min.) prior to exhaust or exhaust air directly to the outside. *Category IC*
 - ix At the completion of the project, terminally clean the work zone according to facility procedures, and install barrier curtains to contain dust and debris before removal of rigid barriers.^{120, 241, 263 - 267} *Category IC*
 - x Flush the water system to clear sediment from pipes. *Category II*
 - xi Restore appropriate ACH, humidity, and pressure differential; clean or replace roughing air filters; dispose spent filters as appropriate. *Category IC*
- 1-2.8 **No recommendation** on routine microbiologic air sampling before, during, or after construction or before or during occupancy of areas housing immunocompromised patients.^{17, 20, 49, 97, 109, 262, 1309} *Unresolved issue*
- 1-2.9 Use an anti-fungal biocide (e.g., copper-8-quinolinolate) in fireproofing materials.^{50, 229, 267, 315} *Category IC*
- 1-2.10 Perform particulate sampling on the HVAC system as part of the commissioning process for newly constructed operating rooms and PE areas.^{100, 120, 277, 293} *Category II*
- 1-2.11 If a case of healthcare-acquired aspergillosis or other opportunistic fungal disease occurs during or immediately after construction, implement appropriate followup measures: *Category IB*
 - 1-2.11.a Ensure that the pressure differentials in the construction zone and in PE are appropriate for those settings *Category IC*

- 1-2.11.b Conduct a prospective search for additional cases and intensify the retrospective review of the hospital's medical and laboratory records.³ *Category IB*
- 1-2.11.c If there is no evidence of ongoing transmission, continue routine maintenance to prevent healthcare-acquired fungal disease.³ *Category IB*
- 1-2.12 If there is evidence of ongoing transmission, conduct an environmental investigation to determine and eliminate the source.^{3, 120, 241, 263 - 267} *Category IB*
 - 1-2.12.a Collect environmental samples from potential sources of airborne fungal spores, especially from sources implicated in the epidemiologic investigation, using preferably a high-volume air sampler rather than settle plates.^{3, 18, 44, 48, 49, 97, 106, 111, 112, 115, 120, 241, 247, 263 - 267, 281, 299} *Category IB*
 - 1-2.12.b If either an environmental source of airborne fungi or an engineering problem with filtration or pressure differentials is identified, promptly perform corrective measures to eliminate the source and route of entry.^{96, 97} *Category IB*
 - 1-2.12.c If an environmental source of airborne fungi is not identified, review infection control measures, including engineering controls, to identify potential areas for correction or improvement. *Category IB*
- 1-2.13 If possible, perform molecular subtyping of *Aspergillus* spp. isolated from patients and the environment to establish strain identities.^{245, 282 - 285} *Category II*
- 1-2.14 If air-supply systems to areas such as PE are not optimal, consider temporary deployment of portable, industrial-grade HEPA filters until rooms with optimal air-handling systems become available.^{3, 120, 263 - 267} *Category II*

1-3. Environmental Infection Control Measures in Special Healthcare Settings

- 1-3.1 Maintain redundant ventilation equipment for emergency replacement in PE, AII, and operating rooms.^{68, 120} *Category IC*
- 1-3.2 Implement infection control measures in care areas for high-risk patients (i.e., patients who require PE rooms, solid organ transplant patients, non-allogeneic neutropenic patients):
 - 1-3.2.a Minimize exposures of these immunocompromised patients to activities that might cause aerosolization of fungal spores (e.g., vacuuming, disruption of ceiling tiles).^{9, 20, 109, 262} *Category IB*
 - 1-3.2.b Incorporate the following dust-control engineering specifications into the planning and construction of new PE units: *Category IC*
 - i Install central or point-of-use HEPA filters for incoming air.^{3, 18, 20, 44, 99 - 104, 120, 247, 304 - 306, 1310, 1311} *Category IB*
 - ii Ensure that rooms are well sealed by: 1) properly constructing windows, doors, and intake and exhaust ports; 2) ensuring that ceilings are smooth and free of fissures, open joints, and crevices; and 3) sealing walls above and below the ceiling.^{3, 111, 120, 305, 306} *Category IC*
 - 1-3.2.c Incorporate the following ventilation engineering specifications into the planning and construction of new PE units: *Category IC*
 - i Ventilate the room to maintain ≥ 12 ACH; place HEPA filters at the room air-supply site.^{3, 9, 120, 234, 305, 306} *Category IC*
 - ii Locate air-intake and exhaust grilles so that clean, filtered air enters from one side of the room, flows across the patient's bed, and exits on the opposite side of the room.^{3, 120, 305, 306} *Category IC*
 - iii Maintain positive room air pressure (2.5 Pa [0.01" water gauge]) in relation to the corridor by supplying air to the room at a rate that is 150 ft³/min. greater than the rate of air exhausted from the room.^{3, 35, 120, 305, 306} *Category IC*
 - iv Maintain airflow patterns and monitor these on a daily basis.²⁶³ *Category IC*
 - v Install self-closing doors in rooms in protective environments.¹²⁰ *Category IC*
 - 1-3.2.d When immunocompromised patients who normally would be in PE require isolation for airborne diseases (e.g., VZV infection, tuberculosis), place these patients in an AII room with an anteroom having an independent exhaust.^{120, 305} *Category IC*
 - 1-3.2.e If an anteroom is not available, place the patient in AII and use portable, industrial-grade HEPA filters to enhance the filtration of spores in the room. *Category II*
 - 1-3.2.f Do not routinely use laminar airflow in PE.^{3, 44, 247, 299, 304 - 306, 1310, 1311} *Category IB*
 - 1-3.2.g Minimize the length of time that immunocompromised patients in PE are outside their rooms for diagnostic procedures and other activities.⁹ *Category IB*

- 1-3.2.h When these patients must leave their rooms, provide them with appropriate respiratory protection:
Category II
- i Equip them with N95 respirators, provided that the patients can tolerate this device, and can be properly fit-tested and trained in its use.^{3, 9, 272} *Category II*
 - ii In the event that the patients cannot tolerate the N95 respirator, use powered air purifying respirators.⁹ *Category II*
 - iii Do not use surgical masks as respiratory protection for these patients.⁹ *Category IB*
- 1-3.3 Implement infection control measures for AII:
- 1-3.3.a Incorporate the following specifications into the planning and construction of new or renovating existing AII units:^{6, 120}
- i Maintain continuous negative air pressure (2.5 Pa [0.01" water gauge]) in relation to the air pressure in the corridor, and monitor air pressure periodically, preferably daily, with audible manometers or smoke tubes at the door.^{305, 306} *Category IB*
 - ii Ensure that rooms are well-sealed by properly constructing windows, doors, and air-intake and exhaust ports.^{305, 306} *Category IC*
 - iii Install self-closing doors.¹²⁰ *Category IC*
 - iv Provide ventilation to ensure ≥ 12 ACH for both renovated rooms and new rooms.^{4, 107, 120} *Category IC*
 - v Direct exhaust air to the outside, away from air-intake and populated areas; if this is not practical, air from the room may be recirculated if passed through a HEPA filter.^{4, 120} *Category IC*
 - vi If supplemental engineering controls for air cleaning are indicated from a risk assessment of the AII area, install UVGI units in the exhaust air ducts of the HVAC system to supplement HEPA filtration or install UVGI fixtures on or near the ceiling to irradiate upper room air.⁴ *Category II*
- 1-3.3.b Use AII rooms for patients with or suspected of having an airborne infection who also require cough-inducing procedures, or use an enclosed booth that is engineered to provide: 1) 12 ACH; 2) negative pressure with an exhaust rate of ≥ 50 ft³/min.; 3) air volume differential set at ≥ 100 ft³/min; and 4) air exhausted directly outside away from air intakes and traffic or exhausted after HEPA filtration prior to recirculation.^{4, 338, 339} *Category IC*
- 1-3.3.c If rooms with these specifications are not available, allow sufficient time to elapse such that 99.9% of airborne contaminants can be removed with ventilation (Appendix B).⁴ *Category IB*
- 1-3.3.d Use negative-pressure rooms with anterooms for patients infected with hemorrhagic fever viruses.²⁰⁰
Category II
- i If an anteroom is not available, use portable, industrial-grade HEPA filters to increase the number of ACH. *Category II*
 - ii Require healthcare workers to wear HEPA respirators when entering the rooms of hemorrhagic fever patients with prominent cough, vomiting, diarrhea, or hemorrhage.²⁰⁰
Category IC
- 1-3.3.e **No recommendation** on negative pressure or isolation for patients with *P. carinii* pneumonia.^{126, 131, 132}
Unresolved issue
- 1-3.4 Implement infection control measures for operating rooms:
- 1-3.4.a Maintain positive-pressure ventilation with respect to corridors and adjacent areas.³⁴⁴ *Category IC*
- 1-3.4.b Maintain a minimum of 15 ACH, with at least 3 ACH of fresh air.¹²⁰ *Category IC*
- 1-3.4.c Filter all recirculated and fresh air through the appropriate filters.¹²⁰ *Category IC*
- 1-3.4.d In rooms not engineered for horizontal laminar airflow, introduce air at the ceiling and exhaust air near the floor.^{345, 346} *Category IC*
- 1-3.4.e Do not use UV lights to prevent surgical-site infections.^{344, 351 - 357} *Category IB*
- 1-3.4.f Keep operating room doors closed except for the passage of equipment, personnel, and patients, and limit entry to essential personnel.³⁴⁰ *Category IC*
- 1-3.4.g Follow appropriate procedures for infectious tuberculosis (TB) patients who also require emergency surgery.^{4, 358} *Category IC*
- i If possible, schedule infectious TB patients as the last surgical cases of the day to maximize the time available for removal of airborne contamination. *Category II*

- ii Use a NIOSH-approved N95 respirator in the operating room.³⁵⁸ *Category IC*
(OSHA; proposed)
- iii Intubate the patient in the operating room, and do not allow the doors to open until 99.9% of the airborne contaminants are removed (Appendix B).⁴ *Category IC*
- iv If the patient is extubated in the operating room, allow adequate time to elapse before the patient and staff leave the suite (Appendix B), or extubate and allow the patient to recover in a AII room negative-pressure room.⁴ *Category IB*
- v Use portable, industrial-grade HEPA filters temporarily if the ACH does not meet the specifications for a negative-pressure area at a minimum, being sure to position the units so that all room air passes through the filter;⁴ provide fresh air as per ventilation standards for operating rooms, as portable units do not meet the requirements for the number of fresh air ACH.^{211, 214} *Category II*

1-3.4.h **No recommendation** for performing orthopedic implant operations in rooms supplied with laminar airflow.^{349, 351} *Unresolved issue*

1-4 Other Aerosol Hazards in Healthcare Facilities

- 1-4.1 In settings where surgical lasers are used, wear appropriate personal protective equipment, including high-filtration surgical masks.^{363, 374} *Category IC*
- 1-4.2 Use central wall suction units with in-line filters to evacuate minimal laser plumes.^{367, 374} *Category IC*
- 1-4.3 Use a mechanical smoke evacuation system with a high-efficiency filter to manage the generation of large amounts of laser plume, or if performing procedures on a patient with extrapulmonary TB.^{4, 367, 374} *Category IC*

2. Water

2-1 Controlling the Spread of Waterborne Microorganisms

- 2-1.1 Practice consistent handwashing/hand hygiene to prevent the spread of waterborne pathogens; use barrier precautions (e.g., gloves) in accordance with current guidelines.^{6, 444, 555, 564, 570} *Category IA*
- 2-1.2 Eliminate contaminated environmental reservoirs (e.g., equipment, solutions) whenever possible.^{444, 445} *Category IB*
- 2-1.3 Clean and disinfect sinks and wash basins on a regular basis. *Category II*
- 2-1.4 Rule out contamination from water whenever waterborne microorganisms, especially NTM, are isolated from clinical specimens collected aseptically from sterile sites or if post-procedural infection or colonization occurs after use of tap water in patient care.^{597 - 599, 604} *Category IB*

2-2 Water Systems in Healthcare Facilities

- 2-2.1 Maintain hot water temperature at the outlet at the highest temperature allowable by state regulations or codes, preferably $\geq 51^{\circ}\text{C}$ ($\geq 124^{\circ}\text{F}$), and maintain cold water temperature at $< 20^{\circ}\text{C}$ ($< 68^{\circ}\text{F}$).^{3, 637} *Category IC*
- 2-2.2 If the hot water temperature can be maintained at $\geq 51^{\circ}\text{C}$ ($\geq 124^{\circ}\text{F}$), install preset thermostatic valves in point-of-use fixtures to help minimize the risk of scalding. *Category II*
- 2-2.3 When state regulations or codes do not allow hot water temperatures $> 43^{\circ}\text{C}$ ($> 110^{\circ}\text{F}$), follow alternative preventive measures to minimize the growth of *Legionella* spp. in water systems. *Category II*
 - 2-2.3.a Periodically increase the hot water temperature to $\geq 66^{\circ}\text{C}$ ($\geq 150^{\circ}\text{F}$) at the point of use (with thermostatic mixing valves present). *Category II*
 - 2-2.3.b Alternatively, if the building cannot be retrofitted with thermostatic mixing valves, chlorinate the water and then flush it through the system.^{637, 678, 679} *Category II*
- 2-2.4 Maintain constant recirculation in hot-water distribution systems serving patient-care areas.¹²⁰ *Category IC*
- 2-2.5 When water disruptions occur, use the following infection control measures:
 - 2-2.5.a Prepare a contingency plan to estimate water demands for the entire facility; *Category IC*
 - 2-2.5.b Post signs advising patients, families, staff, and visitors not to drink tap water until the system is cleared for use by the facility engineer. *Category IC*
 - 2-2.5.c When the system function is restored:
 - i Thoroughly flush the system with water at ambient temperatures, or
 - ii Use high-temperature water flushing or chlorination.⁶³⁷ *Category IC*

- iii Flush and restart equipment and fixtures according to manufacturers' instructions.
Category IC
- 2-2.6 When corrective decontamination of the hot water system is necessary after a disruption in service or a cross-connection with sewer lines has occurred:
 - 2-2.6.a Decontaminate the system when the fewest occupants are present in the building (e.g., nights, weekends).³ *Category II*
 - 2-2.6.b If using high-temperature decontamination, raise the hot-water temperature to 71°C -77°C (160°F - 170°F), progressively flushing each outlet; flush the system for a minimum of 5 minutes.^{3, 637} *Category IC*
 - 2-2.6.c If using chlorination, add enough chlorine, preferably overnight, to achieve a free chlorine residual of ≥ 2 mg/L (≥ 2 ppm) throughout the system.⁶³⁷ *Category IC*
- 2-2.7 Adhere to any "boil water" advisory issued by the municipal water utility. *Category IC*
 - 2-2.7.a Alert patients, families, staff, and visitors not to consume water from drinking fountains, ice, or drinks made from municipal tap water while the advisory is in effect unless the water has been disinfected by bringing to a rolling boil for 1 minute.⁶¹⁹ *Category IC*
 - 2-2.7.b After the advisory is lifted, run faucets and drinking fountains at full flow for several minutes; maintain a high level of surveillance for waterborne disease among patients.⁶¹⁹ *Category IC*
 - 2-2.7.c Change the pre-treatment filter and disinfect the dialysis water system to prevent colonization of the RO membrane and downstream microbial contamination.⁶⁸⁷ *Category IC*
 - 2-2.7.d Run water softeners through a regeneration cycle to restore their capacity and function. *Category IC*
 - 2-2.7.e Inspect water storage tanks to determine if they need to be drained, disinfected, and refilled. *Category IC*
- 2-2.8 Implement procedures to manage a sewage system failure or flooding:
 - 2-2.8.a Develop a contingency plan in accordance with JCAHO requirements.⁶⁸⁶ *Category IC*
 - 2-2.8.b Relocate patients and clean/sterile supplies from affected areas, and close off these areas during clean-up procedures. *Category IC*
 - 2-2.8.c Ensure that the sewage system is fully functional before beginning remediation so that contaminated solids and standing water can be removed. *Category IC*
 - 2-2.8.d If hard-surface equipment, floors, and walls remain in good repair, allow them to dry out; clean with detergent according to standard cleaning procedures. *Category IC*
 - 2-2.8.e Remove absorbent structural items (e.g., carpeting, wallboard, wallpaper) and cloth furnishings if they cannot be easily and thoroughly cleaned and dried within 72 hours; replace with new materials as soon as the underlying structure is declared thoroughly dry by the hospital engineer. *Category IC*
 - 2-2.8.f Clean wood furniture and materials (if still in good repair); allow them to dry thoroughly before restoring varnish or other surface coatings. *Category IC*
 - 2-2.8.g Contain dust and debris during remediation and repair as per recommendation (1-2.7.e). *Category IC*

2-3 Strategies for Preventing Healthcare-Associated Legionellosis

- 2-3.1 Use proper decontamination strategies as needed to eliminate legionellae from the healthcare facility's hot water supply.⁷³⁰ *Category IB*
 - 2-3.1.a When using a pulse (one time) decontamination method, superheat the water by flushing each outlet for ≥ 5 minutes with water at 71°C - 77°C (160°F - 170°F) or hyperchlorinate the system by flushing all outlets for ≥ 5 minutes with water containing ≥ 10 mg/L (≥ 10 ppm) free residual chlorine.^{637, 679, 681, 690, 728} *Category IB*
 - 2-3.1.b After a pulse treatment, maintain water temperatures at the outlet as per the recommendation (2-2.1) wherever practical and permitted by state codes, or chlorinate heated water to achieve 1 - 2 mg/L (1 - 2 ppm) free residual chlorine at the tap.^{26, 436, 637, 677, 692, 693} *Category IC*
 - 2-3.1.c Install preset thermostatic mixing valves in point-of-use fixtures to minimize the risk of scalding or post warning signs at each outlet to alert patients, visitors, and staff about the potential for scalding. *Category IC*
- 2-3.2 **No recommendation** for treating water with ozone, UV light, heavy-metal ions, or monochloramines.^{694 - 710} *Unresolved issue*
- 2-3.3 Implement strategies for preventing Legionnaires' disease.
 - 2-3.3.a Establish a surveillance process to detect healthcare-associated Legionnaires' disease:

- i Inform healthcare personnel (e.g., infection control, physicians, patient-care staff, engineering) about the potential for Legionnaires' disease to occur and measures to prevent and control healthcare-associated legionellosis.^{436, 723} *Category IB*
 - ii Establish mechanisms to provide clinicians with laboratory tests (i.e., culture, urine antigen, DFA serology) for the diagnosis of Legionnaires' disease.^{3, 430} *Category II*
 - iii Maintain a high index of suspicion for healthcare-associated Legionnaires' disease, especially in high-risk patients (e.g., persons with chronic underlying disease, immunocompromised patients, persons ≥ 65 of age).^{3, 377, 385, 416, 422 - 424, 431, 434, 436}
Category II
- 2-3.3.b **No recommendation** on routine culturing of water systems in healthcare facilities that do not have patient-care areas (i.e., transplant units) for persons at high risk for *Legionella* infection.^{26, 434, 675, 677, 681, 711, 717} *Unresolved issue*
- 2-3.3.c If one case of laboratory-confirmed, definite healthcare-associated Legionnaires' disease is identified, OR if two or more cases of laboratory-confirmed, possible healthcare-associated Legionnaires' disease occur during a 6-month period, report the cases to the state and local health departments, and conduct an epidemiologic investigation. *Category II*
 - i If a case occurs in a severely immunocompromised patient, or if severely immunocompromised patients are present in high-risk areas of the hospital (e.g., transplant units), conduct a combined epidemiologic and environmental investigation to determine the source of *Legionella* spp. *Category II*
 - ii If the facility does not treat severely immunocompromised patients, conduct a retrospective review of microbiologic, serologic, and postmortem data to look for previously unidentified cases of healthcare-associated Legionnaires' disease, and begin intensive prospective surveillance for additional cases. *Category II*
- 2-3.3.d If there is no evidence of continued healthcare-associated transmission, continue intensive prospective surveillance for at least 2 months after the initiation of surveillance. *Category II*
- 2-3.3.e If there is evidence of continued healthcare-associated transmission, conduct an environmental investigation to determine the source of *Legionella* spp. *Category IB*
 - i Collect water samples from potential sources of aerosolized water according to the methods described in Appendix C. *Category II*
 - ii Save and subtype isolates of *Legionella* spp. obtained from patients and the environment.^{388 - 396, 727, 728} *Category IB*
- 2-3.3.f If a source is not identified, continue surveillance for new cases for at least 2 months; depending on the scope of the outbreak, either defer decontamination pending identification of the source of *Legionella* spp., or proceed with decontamination of the hospital's water distribution system, with special attention to areas involved in the outbreak. *Category II*
- 2-3.3.g If a source is identified, promptly institute water system decontamination measures as per recommendation (2-3.1).⁷³⁰ *Category IB*
- 2-3.3.h Clean hot-water storage tanks and water heaters to remove accumulated scale and sediment.³⁹⁸
Category II
- 2-3.3.i **No recommendation** for the removal of faucet aerators in areas for immunocompetent patients.
Unresolved issue
- 2-3.4 Assess the efficacy of implemented measures in reducing or eliminating *Legionella* spp. by collecting specimens for culture at 2-week intervals for 3 months. *Category II*
 - 2-3.4.a If *Legionella* spp. are not detected in cultures during 3 months of monitoring at 2-week intervals, collect cultures monthly for another 3 months. *Category II*
 - 2-3.4.b If *Legionella* spp. are detected in one or more cultures, reassess the control measures, modify them accordingly, and repeat the decontamination procedures, with possible approaches including the intensive use of the same technique used for initial decontamination or a combination of superheating and hyperchlorination. *Category II*
- 2-3.5 Keep adequate records of all infection-control measures and environmental test results for potable water systems. *Category II*
- 2-3.6 Strategies for preventing Legionnaires' disease among immunosuppressed patients housed in facilities with transplant units include:

- 2-3.6.a Maintain a high index of suspicion for legionellosis in transplant patients even when environmental surveillance cultures do not yield legionellae.^{429, 430} *Category II*
- 2-3.6.b Depending on state regulations on potable water temperature in public buildings,⁶⁹¹ hospitals housing patients at high risk for healthcare-associated legionellosis should either maintain potable water at the outlet at $\geq 51^{\circ}\text{C}$ [$\geq 124^{\circ}\text{F}$] or $< 20^{\circ}\text{C}$ [$< 68^{\circ}\text{F}$] or chlorinate heated water to achieve 1 - 2 mg/L [1 - 2 ppm] of free residual chlorine at the tap.^{26, 436, 637, 677 - 679, 692, 693} *Category II*
- 2-3.6.c When *Legionella* spp. are not detectable in unit water, remove, clean, and disinfect shower heads and tap aerators in high-risk patient-care areas monthly using a chlorine bleach solution (i.e., 1:100 dilution of bleach).^{637, 709} *Category II*
- 2-3.6.d Facilities with organ transplant units should consider conducting periodic environmental surveillance (culturing) for legionellae in water samples as part of an overall strategy to prevent Legionnaires' disease, the goal of which in these instances should be to maintain water systems with no detectable organisms.^{9, 430} *Category II*
- 2-3.6.e **No recommendation** on the optimal methodology (i.e., frequency or number of sites) for environmental surveillance cultures in transplant units. *Unresolved issue*
- 2-3.6.f If *Legionella* spp. are detected in the water of a transplant unit, the following should be done until *Legionella* spp. are no longer detected by culture:
- i Decontaminate the water supply as per recommendation (2-3.1).^{3, 9, 637} *Category IB*
 - ii Restrict severely immunosuppressed patients from taking showers.^{9, 411} *Category IB*
 - iii Use water that is not contaminated with *Legionella* spp. for HSCT patients' sponge baths.⁹ *Category IB*
 - iv Provide patients with sterile water for tooth brushing, drinking, and for flushing nasogastric tubing during legionellosis outbreaks.^{9, 411} *Category IB*
 - v Remove aerators from faucets and avoid use of tap water from faucets.⁹ *Category II*
- 2-3.6.g Do not use large-volume room air humidifiers that create aerosols (e.g., by Venturi principle, ultrasound, or spinning disk) unless they are subjected to high-level disinfection and filled only with sterile water.^{3, 9} *Category II*

2-4 Cooling Towers and Evaporative Condensers

- 2-4.1 When planning construction of new healthcare facilities, locate cooling towers so that the drift is directed away from the air-intake system, and design the towers to minimize the volume of aerosol drift.^{390, 637, 747} *Category IC*
- 2-4.2 Implement infection control procedures for operational cooling towers.
- 2-4.2.a Install drift eliminators.^{390, 637, 745} *Category IC*
- 2-4.2.b Use an effective biocide on a regular basis. *Category IC*
- 2-4.2.c Maintain towers according to manufacturers' recommendations, and keep detailed maintenance and infection control records, including environmental test results from legionellosis outbreak investigations.⁶³⁷ *Category IC*
- 2-4.2.d If cooling towers or evaporative condensers are implicated in healthcare-associated legionellosis, decontaminate the cooling-tower system.^{390, 391, 747, 748} *Category IB*

2-5 Dialysis Water Quality and Dialysate

- 2-5.1 Adhere to current AAMI standards for quality assurance performance of devices and equipment used to treat, store, and distribute water in hemodialysis centers (both acute and maintenance settings) and for the preparation of concentrates and dialysate.^{749, 750} *Category IA*
- 2-5.2 **No recommendation** on whether more stringent requirements for water quality should be imposed in hemofiltration and hemodiafiltration. *Unresolved issue*
- 2-5.3 Conduct microbiological testing specific to water in dialysis settings. *Category IA*
- 2-5.3.a Perform bacteriologic assays of water and dialysis fluids at least once a month and during outbreaks using standard quantitative methods.^{749, 750, 794, 795} *Category IA*
- i Assay for heterotrophic, mesophilic bacteria.
 - ii Do not use a nutrient-rich media such as blood agar or chocolate agar.
- 2-5.3.b In conjunction with microbiological testing, perform endotoxin testing, especially on samples from water used to reprocess hemodialyzers.^{751, 752, 789} *Category IA*

- 2-5.3.c Ensure that water does not exceed the limits for microbial counts and endotoxin concentrations outlined in Table 26.^{749 - 752} *Category IA*
- 2-5.4 Disinfect water distribution systems in dialysis settings at least weekly.^{749, 750} *Category IA*
- 2-5.5 Design and engineer water systems in dialysis settings so that they are free of joints, dead-end pipes, and unused branches and taps that can harbor bacteria.⁷⁶⁰ *Category IC*
- 2-5.6 Do not use storage tanks in dialysis systems unless they are routinely drained, disinfected, and have an ultrafilter (membrane filter with a pore size sufficient to remove small particles and molecules ≥ 1 kilodalton in size) installed in the water line distal to the storage tank.^{749, 750} *Category IC*

2-6 Ice Machines and Ice

- 2-6.1 Do not handle ice directly by hand, and wash hands before obtaining ice. *Category II*
- 2-6.2 Use a smooth-surface ice scoop to dispense ice; keep the ice scoop on a chain short enough so the scoop cannot touch the floor, or keep the scoop on a clean, hard surface when not in use.^{649, 823} *Category II*
- 2-6.3 Do not store pharmaceuticals or medical solutions on ice intended for consumption; use sterile ice to keep medical solutions cold, or use equipment specifically manufactured for this purpose.^{578, 823} *Category IB*
- 2-6.4 Limit access to ice-storage chests, and keep the container doors closed except when removing ice.⁸²³ *Category II*
- 2-6.5 Clean and disinfect ice-storage chests on a regular basis. *Category II*
 - 2-6.5.a Follow manufacturers' instructions for cleaning and frequency of cleaning. *Category II*
 - 2-6.5.b If instructions are not available, use a general cleaning/disinfecting regimen (Table 29).⁸²³ *Category II*
- 2-6.6 To ensure performance and maintain infection control, perform regular maintenance of ice machines according to manufacturers' instructions or, in the absence of manufacturers' instructions clean, disinfect, and maintain ice machines on a regular basis using a general approach as outlined in Table 30.⁸²³ *Category II*
- 2-6.7 Conduct microbiologic sampling of ice, ice chests, and ice-making machines if indicated for an epidemiologic investigation.^{821 - 823} *Category IB*

2-7 Hydrotherapy Tanks and Pools

- 2-7.1 Drain and clean hydrotherapy equipment (e.g., Hubbard tanks, tubs, whirlpools, jacuzzis, birthing tanks) after each patient, and disinfect equipment surfaces and components in accordance with manufacturers' instructions and relevant scientific literature. *Category IC*
- 2-7.2 In the absence of manufacturers' recommendations for water treatment, add disinfectant to the water:
 - 2-7.2.a Maintain a 15-ppm chlorine residual in the water of small hydrotherapy tanks, Hubbard tanks, and tubs.⁸⁴⁷ *Category II*
 - 2-7.2.b Maintain a 2- to 5-ppm chlorine residual in the water of whirlpools and jacuzzis.⁸⁶³ *Category II*
- 2-7.3 Clean and disinfect hydrotherapy equipment after using tub liners. *Category II*
- 2-7.4 Clean and disinfect inflatable tubs unless they are used as single-use equipment. *Category II*
- 2-7.5 **No recommendation** on the use of antiseptic chemicals (e.g., chloramine-T) in the water during hydrotherapy sessions. *Unresolved issue*
- 2-7.6 Use appropriate infection control measures for large hydrotherapy pools:
 - 2-7.6.a Conduct a risk assessment of patients prior to their use of the pool, deferring patients with draining wounds or fecal incontinence from pool use until their condition resolves. *Category II*
 - 2-7.6.b Use pH and chlorine residual levels appropriate for an indoor pool as provided by local and state health agencies. *Category IC*
- 2-7.7 **No recommendation** on the use in health care of equipment manufactured for home or recreational use. *Unresolved issue*

2-8 Miscellaneous Medical Equipment Connected to Water Systems

- 2-8.1 Clean, disinfect, and maintain automated endoscope reprocessor (AER) equipment according to manufacturers' instructions and relevant scientific literature.^{868 - 870} *Category IB*
 - 2-8.1.a Rinse disinfected endoscopes in water that is bacteria-free at a minimum; if sterile water is not available or practical, use bacteriologically-filtered water (water filtered through 0.1- to 0.2- μ m filters).^{865, 870, 871} *Category IB*
 - 2-8.1.b Flush reprocessed endoscopes with 70% alcohol, followed by forced-air treatment to ensure adequate drying of the internal channels and to prevent the formation of biofilms.^{876, 880, 882} *Category IB*

- 2-8.2 Take precautions to prevent waterborne contamination of dental unit water lines and instruments:
 - 2-8.3.a Flush all dental instruments that use water, including high-speed handpieces for several minutes before the start of each clinic day.⁸⁹² *Category II*
 - 2-8.3.b Ensure that water in dental unit water lines meets nationally recognized drinking water standards (<500 CFU/mL for heterotrophic plate count) at a minimum.^{891 - 893} *Category IC*
 - 2-8.3.c Consult with dental water line manufacturers to determine suitable methods and equipment to obtain good water quality.⁸⁹² *Category II*

3. Environmental Services

3-1 Cleaning and Disinfecting Strategies for Environmental Surfaces in Patient-Care Areas

- 3-1.1 Do not use high-level disinfectants/liquid chemical sterilants on non-critical surfaces for disinfection.^{900, 901} *Category IC*
- 3-1.2 Follow manufacturers' instructions for cleaning and maintaining non-critical medical equipment. *Category II*
- 3-1.3 In the absence of manufacturers' cleaning instructions, follow these procedures:
 - 3-1.3.a Depending on the nature of the surface and the degree of contamination, clean non-critical medical equipment surfaces with a detergent/disinfectant or soap and water, followed with an application of low- to intermediate-level chemical germicide at proper use dilution and for the full contact time required.^{901, 902} *Category II*
 - 3-1.3.b Do not use alcohol to disinfect large surfaces.⁹⁰¹ *Category II*
 - 3-1.3.c Use barrier protective coverings as appropriate for non-critical surfaces that are: 1) touched frequently with gloved hands during the delivery of patient care; 2) likely to become contaminated with blood or body substances; or 3) difficult to clean.⁸⁹² *Category II*
- 3-1.4 Keep housekeeping surfaces (e.g., floors, walls, tabletops) visibly clean on a regular basis and as spills occur. *Category II*
 - 3-1.4.a Use a one-step process and water/detergent or an EPA-registered hospital grade disinfectant/detergent designed for general housekeeping purposes.^{901, 902, 927, 928} *Category II*
 - 3-1.4.b Follow manufacturers' instructions for proper use of cleaning/disinfecting products, paying close attention to specified use dilutions and stated contact times.^{2, 924} *Category II*
 - 3-1.4.c Clean and disinfect high-touch surfaces (e.g., doorknobs, bedrails, light switches, surfaces in and around toilets in patients' rooms) on a more frequent schedule compared to that for minimal touch housekeeping surfaces. *Category II*
 - 3-1.4.d Clean walls, blinds, and window curtains in patient-care areas when they are visibly dusty or soiled.^{2, 915, 916, 923} *Category II*
- 3-1.5 Do not do disinfectant fogging for routine purposes in patient-care areas.² *Category IB*
- 3-1.6 Avoid large-surface cleaning methods that produce mists or aerosols or disperse dust in patient-care areas.^{9, 20, 109, 262} *Category IB*
- 3-1.7 Follow proper procedures for effective use of mops, cloths, and solutions: *Category II*
 - 3-1.7.a Prepare cleaning solutions daily or as needed, and replace with fresh solution frequently according to facility policies and procedures.^{927, 928} *Category II*
 - 3-1.7.b Use clean mops and cloths every time a bucket of cleaning solution is emptied and replenished with clean, fresh solution. *Category II*
 - 3-1.7.c Clean mops and cloths after use and allow to dry before reuse, or, use single-use, disposable mop heads and cloths.^{915, 929 - 931} *Category II*
- 3-1.8 After the last surgical procedure of the day or night, wet vacuum or mop the operating room floors with a single-use mop and an EPA-registered hospital disinfectant.⁷ *Category IB*
- 3-1.9 Do not use tacky mats at the entrance to operating rooms or infection-control suites.⁷ *Category IB*
- 3-1.10 Use proper dusting methods for patient-care areas designated for immunosuppressed patients (e.g., HSCT patients):⁹ *Category IB*
 - 3-1.10.a Wet-dust horizontal surfaces daily using cloths moistened with an EPA-registered hospital disinfectant.^{94, 927} *Category IB*
 - 3-1.10.b Avoid dusting methods that disperse dust (i.e., featherdusting).⁹⁴ *Category IB*
- 3-1.11 Keep vacuums in good repair, and equip vacuums with HEPA filters for use in high-risk patient-care areas.^{9, 94, 927, 936} *Category IB*
- 3-1.12 Close the doors of immunocompromised patients' rooms when vacuuming corridor floors to minimize exposure

- to airborne dust.^{9, 94, 936} *Category IB*
- 3-1.13 Take precautions when using phenolic disinfectants in neonatal units. *Category IB*
- 3-1.13.a Prepare solutions to correct concentrations in accordance with manufacturers' use instructions, or, use pre-mixed formulations.^{937 - 939} *Category IB*
- 3-1.13.b Do not use phenolics to disinfect bassinets or incubators during an infant's stay.^{901, 937 - 939}
Category IB
- 3-1.13.c Rinse phenolic-treated surfaces with water.⁹³⁹ *Category IB*
- 3-2 Cleaning Spills of Blood and Body Substances**
- 3-2.1 Promptly clean and decontaminate spills of blood or other potentially infectious materials.^{911, 940 - 946} *Category IC (OSHA)*
- 3-2.2 Follow proper procedures for site decontamination of spills of blood or blood-containing body fluids.^{940 - 946}
Category IC
- 3-2.2.a Use protective gloves and other personal protective equipment appropriate for this task.⁹¹¹ *Category IC (OSHA)*
- 3-2.2.b If the spill contains large amounts of blood or body fluids, clean the visible matter with disposable absorbent material, and discard the used cleaning materials in appropriate, labeled containment.^{911, 944, 945, 949, 951} *Category IC (OSHA)*
- 3-2.2.c Swab the area with a disposable cloth moderately wetted with disinfectant, and allow the surface to dry.^{911, 949} *Category IC (OSHA)*
- 3-2.3 Use intermediate-level germicides (germicides registered by the EPA for use as hospital disinfectants and labeled tuberculocidal) at recommended dilution and full contact time to decontaminate spills of blood and other body fluids.^{911, 949} *Category IC (OSHA)*
- 3-2.4 Use a one-step cleaning/disinfecting procedure for small spills.^{927, 928} *Category II*
- 3-2.5 If sodium hypochlorite solutions (e.g., household chlorine bleach) are selected for use:
- 3-2.5.a Use a 1:100 dilution (500 ppm available chlorine) to decontaminate nonporous surfaces after cleaning a spill of either blood or body fluids in patient-care settings.^{949, 950} *Category IB*
- 3-2.5.b If a spill involves large amounts of blood or body fluids, or if a blood or culture spill occurs in the laboratory, use a 1:10 dilution (5,000 ppm available chlorine) for the first application of germicide before cleaning.^{902, 949} *Category IB*
- 3-3 Carpeting and Cloth Furnishings**
- 3-3.1 Vacuum carpeting in public areas of healthcare facilities and in general patient-care areas regularly with well-maintained equipment designed to minimize dust dispersion.⁹²⁷ *Category II*
- 3-3.2 Perform a thorough, deep cleaning of carpeting periodically as determined by facility policy using a method that minimizes the production of aerosols and leaves little or no residue.¹¹¹ *Category II*
- 3-3.3 Avoid the use of carpeting in high-traffic zones in patient-care areas or where spills are likely (e.g., burn therapy units, operating rooms, laboratories, intensive care units).^{111, 962, 966} *Category II*
- 3-3.4 Follow proper procedures for managing spills on carpeting. *Category II*
- 3-3.4.a Spot-clean blood or body substance spills promptly.^{911, 949, 950} *Category IC (OSHA)*
- 3-3.4.b If a spill occurs on carpet tiles, replace any tiles contaminated by blood and body fluids or body substances.⁹⁶⁹ *Category IC (OSHA)*
- 3-3.4.c Thoroughly dry or replace wet carpeting within 72 hours to prevent the growth of fungi.⁹ *Category IB*
- 3-3.5 **No recommendation** on the routine use of fungicidal or bactericidal treatments for carpeting in public areas of a healthcare facility or in general patient-care areas. *Unresolved issue*
- 3-3.6 Avoid the use of carpeting in hallways and patient rooms in areas housing immunosuppressed patients (i.e., PE areas).^{9, 111} *Category IB*
- 3-3.7 Avoid the use of upholstered furniture and furnishings in high-risk patient-care areas and in areas with increased potential for body substance contamination (e.g., pediatrics units).⁹ *Category II*
- 3-3.8 **No recommendation** on the use of upholstered furniture and furnishings in general patient-care areas.
Unresolved issue
- 3-4 Flowers and Plants in Patient-Care Areas**
- 3-4.1 Flowers and potted plants need not be restricted from areas for immunocompetent patients.^{495, 670, 977, 979}

Category II

- 3-4.2 Designate the care and maintenance of flowers and potted plants to staff not directly involved with patient care.⁶⁷⁰ *Category II*
- 3-4.3 Do not allow flowers (fresh or dried) or potted plants in patient-care areas for immunosuppressed patients.^{9, 109, 495} *Category II*

3-5 Pest Control

- 3-5.1 Develop pest control strategies, with emphasis on kitchens, cafeterias, laundries, central sterile supply areas, operating rooms, loading docks, and other areas prone to infestations.^{986, 1005} *Category II*
- 3-5.2 Install screens on all windows that open to the outside; keep screens in good repair. *Category II*
- 3-5.3 Contract for routine pest control service by a credentialed pest control specialist who will tailor the application to the needs of a healthcare facility.¹⁰⁰⁵ *Category II*
- 3-5.4 Place laboratory specimens (e.g., fixed sputum smears) in covered containers for overnight storage.^{1000, 1001} *Category II*

3-6 Special Pathogens

- 3-6.1 Develop and maintain cleaning and disinfection procedures to control environmental contamination with antibiotic-resistant gram-positive cocci (e.g., MRSA, VISA, VRE).^{5, 1040 - 1042} *Category IB*
 - 3-6.1.a Pay special attention to cleaning and disinfection of high-touch surfaces in patient-care areas (e.g., bedrails, carts, charts, bedside commodes, bedrails, doorknobs, faucet handles).^{5, 1040 - 1042} *Category IB*
 - 3-6.1.b Ensure compliance by housekeeping staff with cleaning and disinfection procedures.^{5, 1040 - 1042} *Category IB*
 - 3-6.1.c Use chemical germicides appropriate for the surface to be disinfected (e.g., either low- or intermediate level disinfection) for the full contact time and correct use dilution as specified by the manufacturers' instructions.^{1033 - 1037, 1042} *Category IB*
- 3-6.2 Environmental surface culturing can be used to verify the efficacy of hospital policies and procedures before and after cleaning and disinfecting rooms that house patients with VRE.^{5, 1014, 1017, 1018, 1022, 1026} *Category II*
 - 3-6.3.a Prior approval from infection control, in collaboration with the clinical laboratory, must be obtained. *Category II*
 - 3-6.3.b Infection control, with clinical laboratory consultation, must supervise all environmental culturing. *Category II*
- 3-6.3 Develop and maintain cleaning and disinfection procedures to control environmental contamination with *Clostridium difficile*.^{901, 1053, 1063} *Category IB*
 - 3-6.3.a Thoroughly clean and disinfect environmental and medical equipment surfaces on a regular basis using disinfectants at proper use dilutions and full contact time.^{901, 1053, 1063} *Category IB*
 - 3-6.3.b Use appropriate hand hygiene or handwashing, personal protective equipment (e.g., gloves), and isolation precautions during cleaning and disinfecting procedures.^{901, 1053} *Category IB*
- 3-6.4 Advise families, visitors, and patients about the importance of handwashing or hand hygiene to minimize the spread of fecal contamination to surfaces.⁹⁰¹ *Category II*
- 3-6.5 Do not use high-level disinfectants (liquid chemical sterilants) on environmental surfaces.^{2, 900, 901} *Category IC*
- 3-6.6 **No recommendation** on the use of specific low- or intermediate-level disinfectants with respect to environmental control of *C. difficile*. *Unresolved issue*
- 3-6.7 Develop and maintain cleaning and disinfection procedures to control environmental contamination with respiratory and enteric viruses in pediatric-care units. *Category II*
 - 3-6.7.a Clean surfaces that have been contaminated with body substances; disinfect cleaned surfaces with an intermediate-level disinfectant at proper use dilution and contact time.¹⁰⁷⁵ *Category II*
 - 3-6.7.b Use disposable barrier coverings as appropriate to minimize surface contamination. *Category II*
- 3-6.8 Develop and maintain cleaning and disinfection procedures to control environmental contamination with Creutzfeldt-Jakob disease (CJD) agent in patient-care areas. *Category II*
 - 3-6.8.a In the absence of contamination with central nervous system tissue, extraordinary measures (e.g., use of 2N sodium hydroxide [NaOH] or full-strength sodium hypochlorite [chlorine bleach]) are not needed for routine cleaning or terminal disinfection of a room housing a known or suspected CJD patient. *Category II*
 - 3-6.8.b Use standard procedures for containment, cleaning, and decontamination of blood spills on surfaces as

- previously described (3-2). *Category II*
- 3-6.8.c Use: 1) a sodium hypochlorite solution of $\geq 20,000$ ppm [1:2 dilution] for 2 hour contact time; or 2) 1N NaOH for 2 hours contact time; or 3) 2N NaOH for 1 hour contact time to decontaminate operating room or autopsy surfaces with central nervous system or cerebral spinal fluid contamination from a known or suspected CJD patient.^{1086, 1104, 1113, 1120} *Category II*
- 3-6.4.d Use disposable, impervious covers to minimize body substance contamination to autopsy tables and surfaces. *Category II*

4. Environmental Sampling

4-1 General Recommendations

- 4-1.1 Do not conduct random, undirected microbiologic sampling of air, water, and environmental surfaces in healthcare facilities.^{2, 1127} *Category IB*
- 4-1.2 When indicated, conduct microbiologic sampling as part of an epidemiologic investigation.^{2, 1127} *Category IB*
- 4-1.3 Limit microbiologic sampling for quality assurance purposes to: 1) biological monitoring of sterilization processes; 2) monthly cultures of water and dialysate in hemodialysis units; and 3) short-term evaluation of the impact of infection control measures or changes in infection control protocols.^{2, 1127} *Category IB*

4-2 Air, Water, and Environmental Surface Sampling

- 4-2.1 Select a high-volume air sampling device if anticipated levels of microbial airborne contamination are expected to be low.^{278, 1131, 1136, 1137} *Category II*
- 4-2.2 Do not use settle plates to quantify the concentration of airborne fungal spores.²⁷⁸ *Category II*
- 4-2.3 When sampling water, choose growth media and incubation conditions that will facilitate the recovery of waterborne organisms.⁸⁹⁵ *Category II*
- 4-2.4 When using a sample/rinse method for sampling an environmental surface, develop and document a procedure for manipulating the swab, gauze, or sponge in a reproducible manner so that results are comparable.¹¹⁵¹ *Category II*
- 4-2.5 When environmental samples and patient specimens are available for comparison, perform the laboratory analysis on the recovered microorganisms down to the species level at a minimum and beyond the species level if possible.¹¹²⁷ *Category II*
- 4-2.6 When conducting any form of environmental sampling, fully document departures from standard methods.^{895, 1127, 1136, 1137, 1151} *Category II*

5. Laundry and Bedding

5-1 Healthcare Worker Issues

- 5-1.1 Employers must launder workers' personal protective garments or uniforms that are contaminated with blood or other potentially infectious materials.⁹¹¹ *Category IC (OSHA)*
- 5-1.2 **No recommendation** on home laundering of uniforms or apparel not considered personal protective equipment if these garments are not soiled with blood or other potentially infectious material. *Unresolved issue*

5-2 Laundry Facilities and Equipment

- 5-2.1 Maintain the receiving area for soiled linens at negative pressure compared to the clean areas of the laundry.¹¹⁷⁰⁻¹¹⁷² *Category IC*
- 5-2.2 Ensure that laundry areas have handwashing facilities and products and appropriate personal protective equipment available for workers.⁹¹¹ *Category IC (OSHA)*
- 5-2.3 Use and maintain laundry equipment according to manufacturers' instructions.^{1164, 1173} *Category II*
- 5-2.4 Do not leave damp textiles or fabrics in machines overnight.¹¹⁶⁴ *Category II*
- 5-2.5 Disinfection of washing and drying machines in residential care is not needed as long as gross soil is removed before washing and proper washing and drying procedures are used. *Category II*

5-3 Routine Handling of Soiled Laundry

- 5-3.1 Handle soiled textiles and fabrics with minimum agitation to avoid contamination of air, surfaces, and persons.^{6, 911, 1169} *Category IC (OSHA)*
- 5-3.2 Bag or otherwise contain soiled textiles and fabrics at the point of use. *Category IC*
- 5-3.2.a Do not sort or pre-rinse soiled textiles or fabrics in patient-care areas.⁹¹¹ *Category IC (OSHA)*

- 5-3.2.b Use leak-resistant containment for textiles and fabrics soiled with blood or body substances.^{911, 1168}
Category IC (OSHA)
- 5-3.2.c Identify bags or containers for soiled linens with labels, color coding, or other alternative means of communication as appropriate.⁹¹¹ *Category IC (OSHA)*
- 5-3.2.d Do not double-bag soiled textiles and fabrics.¹¹⁷⁴ *Category II*
- 5-3.3 If laundry chutes are used, ensure that they are properly designed and maintained; toss bagged laundry into chutes as opposed to loose items.^{1177 - 1180} *Category IC*
- 5-3.4 Establish a facility policy to determine when textiles or fabrics should be sorted in the laundry facility (i.e., before or after washing).^{1181, 1182} *Category II*
- 5-4 Laundry Process**
- 5-4.1 If hot-water laundry cycles are used, wash with detergent in water at least 71°C (160°F) for at least 25 minutes.^{2, 120} *Category IC*
- 5-4.2 **No recommendation** on a hot-water temperature setting and cycle duration for items laundered in residence-style healthcare facilities. *Unresolved issue*
- 5-4.3 Follow fabric-care instructions and special laundering requirements for items used in the facility.¹¹⁸⁷ *Category II*
- 5-4.4 If low-temperature (<70°C [$<160^{\circ}\text{F}$]) laundry cycles are used, use chemicals suitable for low-temperature washing at proper use concentration.^{1161, 1190 - 1194} *Category II*
- 5-4.5 Package, transport, and store clean textiles and fabrics by methods that will ensure their cleanliness and protect them from dust and soil during interfacility loading, transport, and unloading.² *Category IC*
- 5-5 Microbiological Sampling of Linens**
- 5-5.1 Do not conduct routine microbiological sampling of clean linens.^{2, 1195} *Category IB*
- 5-5.2 Use microbiological sampling during outbreak investigations if epidemiologic evidence suggests a role for healthcare linens and clothing in disease transmission.¹¹⁹⁵ *Category IB*
- 5-6 Special Laundry Situations**
- 5-6.1 Use sterilized linens, drapes, and gowns for situations requiring sterility in the patient-care field.⁷ *Category IB*
- 5-6.2 Use hygienically clean linens in neonatal intensive care units.^{939, 1197} *Category IB*
- 5-6.3 Follow manufacturers' recommendations for cleaning fabric items with rubber backing. *Category II*
- 5-6.4 Do not use dry cleaning for routine laundering in healthcare facilities.^{1198 - 1200} *Category II*
- 5-6.5 Evaluate label claims on antimicrobial mattresses, linens, and clothing before using these to replace standard bedding and other fabric items.¹²¹¹ *Category II*
- 5-6.6 **No recommendation** on using disposable fabrics and textiles versus durable goods. *Unresolved issue*
- 5-7 Mattresses and Pillows**
- 5-7.1 Keep mattresses dry; discard them if they become and remain wet or stained, particularly in burn units.^{1214 - 1219} *Category IB*
- 5-7.2 Maintain mattress cover integrity. *Category IB*
- 5-7.2.a Clean and disinfect mattress covers using disinfectants that are compatible with the cover materials to prevent the development of tears, cracks, or holes in the cover.^{1214 - 1219} *Category IB*
- 5-7.2.b Replace mattress covers if they become torn or otherwise in need of repair. *Category II*
- 5-7.2.c Do not stick needles into the mattress through the cover. *Category II*
- 5-7.3 Change mattress covers between patients.^{1214 - 1219} *Category IB*
- 5-7.4 Launder pillow covers and washable pillows in the hot-water cycle.¹²¹⁹ *Category IB*
- 5-8 Air-Fluidized Beds**
- 5-8.1 Follow manufacturers' instructions for bed maintenance and decontamination. *Category II*
- 5-8.2 Change the polyester filter sheet at least weekly or as indicated by the manufacturer.^{1221, 1222, 1226, 1227} *Category II*
- 5-8.3 Clean and disinfect the polyester filter sheet thoroughly, especially between patients.^{1221, 1222, 1226, 1227} *Category IB*
- 5-8.4 Consult the facility engineer to determine the proper location of air-fluidized beds in negative-pressure rooms.¹²³⁰ *Category IC*

6. Animals in Healthcare Facilities

6-1 General Infection Control Measures for Human/Animal Encounters in Health Care

- 6-1.1 Minimize contact with animal saliva, dander, urine, and feces.^{1265 - 1267} *Category II*
- 6-1.2 Wash hands or use an alcohol hand gel after any animal contact.^{8, 1252} *Category II*

6-2 Pet Visitation, Pet Therapy, and Resident Animal Programs

- 6-2.1 Enroll animals that are fully vaccinated, healthy, clean, well-groomed, and negative for enteric pathogens or otherwise have completed recent antihelminthic treatment.¹²⁵² *Category II*
- 6-2.2 Enroll dogs that are trained with the assistance or under the direction of individuals who are experienced in this field. *Category II*
- 6-2.3 Ensure that animals are supervised by persons who know the animals' behavior and can control them.¹²⁵² *Category II*
- 6-2.4 Conduct pet therapy sessions in a central or public area of the facility.¹²⁵² *Category II*
- 6-2.5 Take precautions to mitigate allergic responses to animals. *Category II*
 - 6-2.5.a Minimize shedding of animal dander by bathing animals within 2 days of a visit.¹²⁶¹ *Category II*
 - 6-2.5.b Groom animals to remove loose hair before a visit, or using a therapy animal cape.¹²⁶¹ *Category II*
- 6-2.6 Use routine cleaning protocols for housekeeping surfaces after therapy sessions. *Category II*
- 6-2.7 Restrict resident animals from access to patient-care areas, food preparation areas, places where people are eating, laundry, central sterile supply areas, sterile and clean supply storage areas, medication preparation areas, operating rooms, isolation areas, and protective environments. *Category II*

6-3 Protective Measures for Immunocompromised Patients

- 6-3.1 Advise patients to avoid contact with animal feces and body fluids such as saliva, urine, or solid litter box material.⁸ *Category II*
- 6-3.2 Promptly clean and treat scratches, bites, or other accidents that break the skin.⁸ *Category II*
- 6-3.3 Advise patients to avoid direct or indirect contact with reptiles.¹²⁴⁴ *Category IB*
- 6-3.4 Do not conduct pet visitation or pet therapy programs in areas where immunocompromised patients receive care.¹²⁵² *Category IB*
- 6-3.5 **No recommendation** on permitting pet visits to terminally ill immunosuppressed patients outside their protective environment. *Unresolved issue*

6-4 Service Animals

- 6-4.1 Avoid the use of nonhuman primates and reptiles as service animals. *Category II*
- 6-4.2 Allow service animals access to the facility in accordance with the Americans with Disabilities Act of 1990, unless the presence of the animal creates a direct threat to other persons or interferes with the provision of goods and services.^{1266, 1273} *Category IC (Justice)*
- 6-4.3 Restrict service animals from entering areas that normally require additional precautions to prevent disease transmission (e.g., isolation areas, protective environments, operating rooms, intensive care units, burn therapy units).¹²⁶⁶ *Category II*
- 6-4.4 If a service animal must be separated from its handler, designate a responsible person to supervise the animal, or have a crate or carrier available to contain the animal temporarily.¹²⁶⁶ *Category II*

6-5 Animals as Patients in Human Healthcare Facilities

- 6-5.1 If animals must be brought into human healthcare facilities for care, avoid the use of operating rooms or other patient-care areas where invasive procedures are performed (e.g., cardiac catheterization laboratories, invasive nuclear medicine areas). *Category II*
- 6-5.2 Clean and disinfect or sterilize equipment that has been in contact with animals, or use disposable equipment. *Category II*
- 6-5.3 If reusable medical or surgical instruments are used in an animal procedure, restrict future use of these instruments to animals only. *Category II*

6-6 Research Animals in Healthcare Facilities

- 6-6.1 Use animals obtained from quality stock, or quarantine incoming animals to detect zoonotic diseases. *Category II*

- 6-6.2 Treat sick animals, or remove them from the facility. *Category II*
- 6-6.3 Provide prophylactic vaccinations, as available, to animal handlers and high-risk contacts. *Category II*
- 6-6.4 Ensure proper ventilation through appropriate facility design and location.¹²⁷⁷ *Category IC (USDA)*
 - 6-6.4.a Keep animal rooms at negative pressure relative to corridors. *Category IC*
 - 6-6.4.b Prevent air in animal rooms from recirculating elsewhere in the healthcare facility. *Category IC*
- 6-6.5 Restrict access to animal facilities to essential personnel. *Category II*
- 6-6.6 Establish employee occupational health programs specific for the animal research facility, and coordinate management of post-exposure procedures specific for zoonoses with occupational health clinics in the healthcare facility.⁹⁵² *Category IB*
- 6-6.7 Document standard operating procedures for the unit.⁹⁵² *Category IC*
- 6-6.8 Conduct routine employee training.⁹⁵² *Category II*
- 6-6.9 Use precautions to prevent the development of asthma in animal workers.⁹⁵² *Category II*

7. Regulated Medical Waste

7-1 Categories of Regulated Medical Waste

- 7-1.1 Designate the following as major categories of medical waste that require special handling and disposal precautions: 1) microbiology laboratory wastes [e.g., cultures and stocks of microorganisms]; 2) bulk blood, blood products, blood and body fluid specimens; 3) pathology and anatomy waste; and 4) sharps [e.g., needles, scalpels].² *Category II*
- 7-1.2 Consult federal, state, and local regulations to determine if other waste items are considered regulated medical wastes. *Category IC (EPA, DHHS, States)*

7-2 Disposal Plan for Regulated Medical Wastes

- 7-2.1 Develop a plan for the collection, handling, pre-disposal treatment, and terminal disposal of regulated medical wastes. *Category IC (EPA, States)*
- 7-2.2 Designate a person or persons to be responsible for establishing, monitoring, reviewing, and administering the plan. *Category II*

7-3 Handling, Transport, and Storage of Regulated Medical Wastes

- 7-3.1 Inform personnel involved in the handling and disposal of potentially infective waste of the possible health and safety hazards; ensure that they are trained in appropriate handling and disposal methods.⁹¹¹ *Category IC (OSHA)*
- 7-3.2 Manage the handling and disposal of regulated medical wastes generated in isolation areas using the same methods as for similar wastes from other patient-care areas.² *Category II*
- 7-3.3 Use proper sharps disposal strategies. *Category IC*
 - 7-3.3.a Use a sharps container capable of maintaining its impermeability after waste treatment (e.g., autoclaving) to avoid subsequent physical injuries during final disposal.⁹¹¹ *Category IC (OSHA)*
 - 7-3.3.b Place disposable syringes with needles (including sterile sharps that are being discarded), scalpel blades, and other sharp items into puncture-resistant containers located as close as practical to the point of use.⁹¹¹ *Category IC*
 - 7-3.3.c Do not bend, recap, or break used syringe needles before discarding them into a container.^{6, 1294} *Category II*
- 7-3.4 Store regulated medical wastes awaiting treatment in a properly ventilated area that is inaccessible to vertebrate pests; use waste containers that prevent the development of noxious odors. *Category IC (States)*
- 7-3.5 If treatment options are not available at the site where the medical waste is generated, transport regulated medical wastes in closed, impervious containers to the on-site treatment location or to another facility for treatment as appropriate. *Category IC (States)*

7-4 Treatment and Disposal of Regulated Medical Wastes

- 7-4.1 Treat regulated medical wastes using a method approved by the state (e.g., steam sterilization, incineration, interment, or an alternative treatment technology) before disposal in a sanitary landfill. *Category IC (States)*
- 7-4.2 Follow precautions for treating microbiological wastes (e.g., amplified cultures and stocks of microorganisms). *Category II*
 - 7-4.2.a Biosafety level 4 laboratories must autoclave microbiological wastes in the laboratory before transport to

and disposal in a sanitary landfill.⁹⁵² *Category II*

7-4.2.b Biosafety level 3 laboratories must autoclave microbiological wastes in the laboratory or incinerate them at the facility before transport to and disposal in a sanitary landfill.⁹⁵² *Category II*

7-4.2.c Biosafety levels 1 and 2 laboratories should develop strategies to reinstate the capacity of autoclaving amplified microbial cultures and stocks on-site instead of packaging and shipping untreated wastes to an off-site facility for treatment and disposal.^{952, 1298 - 1300} *Category II*

7-4.3 Laboratories that isolate select agents from clinical specimens must comply with federal regulations for the receipt, transfer, and management (including proper disposal) of these agents.¹²⁹² *Category IC* (DHHS)

7-4.4 Sanitary sewers may be used for the safe disposal of blood, suctioned fluids, ground tissues, excretions, and secretions, provided that local sewage discharge requirements are met and that the state has declared this to be an acceptable method of disposal.¹²⁹³ *Category II*

7-5 Special Precautions for Wastes Generated during Care of Patients with Rare Diseases

7-5.1 When discarding items contaminated with blood and body fluids from viral hemorrhagic fever patients, use methods that minimize the production of aerosols.^{6, 200} *Category II*

7-5.2 Manage properly contained wastes from areas providing care to viral hemorrhagic fever patients in accordance with recommendations for other isolation areas (7-3.2).^{2, 6} *Category II*

7-5.3 Decontaminate bulk blood and body fluids from viral hemorrhagic fever patients using either autoclaving or chemical treatment before disposal.⁶ *Category II*

7-5.4 Follow the recommendation for managing isolation room medical wastes when disposing waste from clinical areas providing care for CJD patients (7-3.2). *Category II*

III. References

1. Simmons BP. Guideline for hospital environmental control. Atlanta GA: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control; 1981.
2. Garner JS, Favero MS. Guideline for handwashing and hospital environmental control. Atlanta GA: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control; 1985. Also *Infect Control* 1986; 7: 231-243.
3. Centers for Disease Control and Prevention. Guidelines for prevention of nosocomial pneumonia. *MMWR* 1997; 46 (RR-1): 1-79.
4. Centers for Disease Control and Prevention. Guidelines for preventing the transmission of *Mycobacterium tuberculosis* in health-care facilities. *MMWR* 1994; 43 (RR-13): 1-132.
5. Centers for Disease Control and Prevention. Recommendations for preventing the spread of vancomycin resistance. Recommendations of the Hospital Infection Control Practices Advisory Committee (HICPAC). *MMWR* 1995; 44 (RR-12): 1-13.
6. Garner JS, Hospital Infection Control Practices Advisory Committee. Guideline for isolation precautions in hospitals. *Infect Control Hosp Epidemiol* 1996; 17: 53-80.
7. Mangram AJ, Horan TC, Pearson ML, Silver LC, Jarvis WR, Hospital Infection Control Practices Advisory Committee. Guideline for prevention of surgical site infection, 1999. *Infect Control Hosp Epidemiol* 1999; 20: 247-280.
8. Centers for Disease Control and Prevention. USPHS/IDSA guidelines for the prevention of opportunistic infections in persons infected with human immunodeficiency virus. *MMWR* 1999; 48 (RR-10): 1-66.
9. Centers for Disease Control and Prevention. CDC/IDSA/ASBMT guidelines for the prevention of opportunistic infections in hematopoietic stem cell transplant recipients. *MMWR* 2000; 49 (RR-10): 1-128.
10. Garner JS. The CDC Hospital Infection Control Practice Advisory Committee. *Am J Infect Control* 1993; 21: 160-162.
11. Rhame FS. The inanimate environment. In: *Hospital Infections*, 4th Ed. Bennett JV, Brachman PS, eds. Philadelphia PA; Lippincott-Raven; 1998: p. 299-324.
12. Weber DJ, Rutala WA. Environmental issues and nosocomial infections. In: *Prevention and Control of Nosocomial Infections*, 3rd Ed. Wenzel RP, ed. Baltimore MD; Williams & Wilkins; 1997: p. 491-514.
13. Greene VW. Microbiological contamination control in hospitals. *Hospitals JAHA* 1969; 43: 78-88.
14. American Hospital Association. *Hospital Statistics*, 2000 Ed.; Table 1: Historical trends in utilization, personnel, and finances for selected years from 1946 through 1998. Chicago IL; Health Forum LLC; 2000: p. 2-3.
15. McKee B. Neither bust nor boom. *Architecture* 1998;
<http://www.britannica.com/bcom/magazine/article/0,5744,39579,00.html>
16. Croswell CL. Better, not bigger: Construction costs soar on wings of patient demand, Construction and Design survey finds. *Mod Healthc* 1999; 29 (12): 23-26, 28-34, 36-38.
17. Sarubbi FA Jr, Kopf BB, Wilson NO, McGinnis MR, Rutala WA. Increased recovery of *Aspergillus flavus* from respiratory specimens during hospital construction. *Am Rev Respir Dis* 1982; 125: 33-38.
18. Arnow PM, Sadigh MC, Weil D, Chudy R. Endemic and epidemic aspergillosis associated with in-hospital replication of *Aspergillus* organisms. *J Infect Dis* 1991; 164: 998-1002.
19. Flynn PM, Williams BG, Hethrington SV, Williams BF, Giannini MA, Pearson TA. *Aspergillus terreus* during hospital renovation. *Infect Control Hosp Epidemiol* 1993; 14: 363-365.
20. Weems JJ Jr, Davis BJ, Tablan OC, Kaufman L, Martone WJ. Construction activity: An independent risk factor for invasive aspergillosis and zygomycosis in patients with hematologic malignancy. *Infect Control* 1987; 8: 71-75.
21. Streifel AJ, Stevens PP, Rhame FS. In-hospital source of airborne *Penicillium* species spores. *J Clin Microbiol* 1987; 25: 1-4.
22. Noskin GA, Stosor V, Cooper J, Peterson L. Recovery of vancomycin-resistant enterococci on fingertips and environmental surfaces. *Infect Control Hosp Epidemiol* 1995; 16: 577-581.
23. Manian FA, Meyer L, Jenne J. *Clostridium difficile* contamination of blood pressure cuffs: A call for a closer look at gloving practices in the era of universal precautions. *Infect Control Hosp Epidemiol* 1996; 17: 180-182.
24. McFarland LV, Mulligan NE, Kwok RYY, et al. Nosocomial acquisition of *Clostridium difficile* infection. *N Engl J Med* 1989; 320: 204-210.
25. Nath SK, Thomely JH, Kelly M, et al. A sustained outbreak of *Clostridium difficile* in a general hospital: Persistence of a toxigenic clone in four units. *Infect Control Hosp Epidemiol* 1994; 15: 382-389.
26. Johnson JT, Yu VL, Best MG, et al. Nosocomial legionellosis in surgical patients with head and neck cancer:

Implications for epidemiological reservoir and mode of transmission. *Lancet* 1985; 2: 298-300.

27. Blatt SP, Parkinson MD, Pace E, et al. Nosocomial Legionnaires' disease: Aspiration as a primary mode of disease acquisition. *Am J Med* 1993; 95: 16-22.
28. Bert F, Maubec E, Bruneau B, Berry P, Lambert-Zechovsky N. Multi-resistant *Pseudomonas aeruginosa* associated with contaminated tap-water in a neurosurgery intensive care unit. *J Hosp Infect* 1998; 39: 53-62.
29. Muyldermans G, de Smet F, Perrard D, et al. Neonatal infections with *Pseudomonas aeruginosa* associated with a water-bath used to thaw fresh frozen plasma. *J Hosp Infect* 1998; 39: 309-314.
30. BATTERY JP, Alabaster SJ, Heine FG, et al. Multi-resistant *Pseudomonas aeruginosa* outbreak in a pediatric oncology ward related to bath toys. *Pediatr Infect Dis J* 1998; 17: 509-513.
31. Bolan G, Reingold AL, Carson LA, et al. Infections with *Mycobacterium chelonae* in patients receiving dialysis and using processed hemodialyzers. *J Infect Dis* 1985; 152: 1013-1019.
32. Lowry PW, Beck-Sague CM, Bland LA, et al. *Mycobacterium chelonae* infection among patients receiving high-flux dialysis in a hemodialysis clinic in California. *J Infect Dis* 1990; 161: 85-90.
33. Schaal KP. Medical and microbiological problems arising from airborne infection in hospitals. *J Hosp Infect* 1991; 18 (Suppl A): 451-459.
34. Osterholm MT, Hedberg CW, Moore KA. Epidemiology of infectious diseases. In: *Principles and Practice of Infectious Diseases*, 5th Ed.; Mandell GL, Bennett JE, Dolin R, eds. Philadelphia PA; Churchill Livingstone; 2000: p. 156-167.
35. Streifel AJ. Design and maintenance of hospital ventilation systems and prevention of airborne nosocomial infections. In: *Hospital Epidemiology and Infection Control*, 2nd Ed.; Mayhall CG, ed. Philadelphia PA; Lippincott Williams & Wilkins; 1999: p. 1211-1221.
36. Bodey GP, Vartivarian S. Aspergillosis. *Eur J Clin Microbiol Infect Dis* 1989; 8: 413-437.
37. Latgé JP. *Aspergillus fumigatus* and aspergillosis. *Clin Microbiol Rev* 1999; 12: 310-350.
38. Derouin F. Special issue on aspergillosis. *Pathol Biol* 1990; 42: 625-736.
39. Dixon DD, Walsh TJ. Human pathogenesis. In: *Aspergillus, Biology and Industrial Application*; Bennett JW, Klich MA, eds. Boston MA; Butterworth-Heinemann; 1992: p. 249-267.
40. Kurup VP, Kumar A. Immunodiagnosis of aspergillosis. *Clin Microbiol Rev* 1991; 4: 439-456.
41. Latgé JP, Paris S, Sarfati J, Debeauvais JP, Beauvais A, Jatton-Ogay K, Monod M. Infectivity of *Aspergillus fumigatus*. In: *Host-Fungus Interplay*; Vanden Bossche H, Stevens DA, Odds FC, eds. Bethesda MD; National Foundation for Infectious Diseases; 1997: p. 99-110.
42. Schaffner A. Host defense in aspergillosis. In: *New Strategies in Fungal Disease*; Bennett E, Hay RJ, Peterson PK, eds. Edinburgh United Kingdom; Churchill Livingstone; 1992: p. 98-112.
43. Vanden Bossche H, Mackenzie DWR, Cauwenbergh G, eds. *Aspergillus and Aspergillosis*. New York NY; Plenum Press; 1988.
44. Sherertz RJ, Belani A, Kramer BS, et al. Impact of air filtration on nosocomial *Aspergillus* infections: Unique risk of bone marrow transplant recipients. *Am J Med* 1987; 83: 709-718.
45. Young RC, Bennett JE, Vogel CL, Carbone PP, DeVita VT. Aspergillosis: The spectrum of the disease in 98 patients. *Medicine* 1970; 49: 147-173.
46. Rhame FS. Lessons from the Roswell Park bone marrow transplant aspergillosis outbreak. *Infect Control* 1985; 6: 345-346.
47. Rotsein C, Cummings KM, Tidings J, et al. An outbreak of invasive aspergillosis among allogeneic bone marrow transplants: A case-control study. *Infect Control* 1985; 6: 347-355.
48. Aisner J, Schimpff SC, Bennett JE, Young VM, Wiernik PH. *Aspergillus* infections in cancer patients: Association with fireproofing materials in a new hospital. *JAMA* 1976; 235: 411-412.
49. Arnow PM, Anderson RI, Mainous PD, Smith EJ. Pulmonary aspergillosis during hospital renovation. *Am Rev Respir Dis* 1978; 118: 49-53.
50. Streifel AJ, Lauer JL, Vesley D, Juni B, Rhame FS. *Aspergillus fumigatus* and other thermotolerant fungi generated by hospital building demolition. *Appl Environ Microbiol* 1983; 46: 375-378.
51. Hopkins CC, Weber DJ, Rubin RH. Invasive aspergillosis infection: Possible non-ward common source within the hospital environment. *J Hosp Infect* 1989; 13: 19-25.
52. Denning DW. Invasive aspergillosis. *Clin Infect Dis* 1998; 26: 781-805.
53. Manuel RJ, Kibbler CC. The epidemiology and prevention of invasive aspergillosis. *J Hosp Infect* 1998; 39: 95-109.
54. Kennedy HF, Simpson EM, Wilson N, Richardson MD, Michie JR. *Aspergillus flavus* endocarditis in a child with

neuroblastoma. *J Infect* 1998; 36: 126-127.

55. McCarty JM, Flam MS, Pullen G, Jones R, Kassel SH. Outbreak of primary cutaneous aspergillosis related to intravenous arm boards. *J Pediatr* 1986; 108 (Pt.1): 721-724.

56. Goldberg B, Eversmann WW, Eitzen EM Jr. Invasive aspergillosis of the hand. *J Hand Surg* 1982; 7: 38-42.

57. Grossman ME, Fithian EC, Behrens C, Bissinger J, Fracaro M, Neu HC. Primary cutaneous aspergillosis in six leukemic children. *J Am Acad Dermatol* 1985; 12: 313-318.

58. Panke TW, McManus AT, Spebar MJ. Infection of a burn wound by *Aspergillus niger*: Gross appearance simulating ecthyma gangrenosa. *Am J Clin Pathol* 1979; 72: 230-232.

59. Fraser DW, Ward JL, Ajello L, Plikaytis BD. Aspergillosis and other systemic mycoses: The growing problem. *JAMA* 1979; 242: 1631-1635.

60. Iwen PC, Reed EC, Armitage JO, et al. Nosocomial invasive aspergillosis in lymphoma patients treated with bone marrow or peripheral stem cell transplants. *Infect Control Hosp Epidemiol* 1993; 14: 131-139.

61. Cordonnier C, Bernaudin JF, Bierling P, Huet Y, Vernant JP. Pulmonary complications occurring after allogeneic bone marrow transplantation: A study of 130 consecutively transplanted patients. *Cancer* 1986; 58: 1047-1054.

62. Klimowski LL, Rotstein C, Cummings KM. Incidence of nosocomial aspergillosis in patients with leukemia over a twenty-year period. *Infect Control Hosp Epidemiol* 1989; 10: 299-305.

63. Walmsley S, Devi S, King S, Schneider R, Richardson S, Ford-Jones L. Invasive *Aspergillus* infections in a pediatric hospital: A ten-year review. *Pediatr Infect Dis* 1993; 12: 673-682.

64. Pannuti CS, Pfaller MA, Wenzel RP. Nosocomial pneumonia in adult patients undergoing bone marrow transplantation: A 9-year study. *J Clin Oncol* 1991; 9: 77-84.

65. Wingard JR, Beals SU, Santos GW, Mertz WG, Saral R. *Aspergillus* infections in bone marrow transplant recipients. *Bone Marrow Transplant* 1987; 2: 175-181.

66. Humphreys H, Johnson EM, Warnock DW, Willatts SM, Winter RJ, Speller DCE. An outbreak of aspergillosis in a general ITU. *J Hosp Infect* 1991; 18: 167-177.

67. Sessa A, Meroni M, Battini G, et al. Nosocomial outbreak of *Aspergillus fumigatus* infection among patients in a renal unit? *Nephrol Dial Transplant* 1996; 11: 1322-1324.

68. Anderson K, Morris G, Kennedy H, et al. Aspergillosis in immunocompromised pediatric patients: Associations with building hygiene, design, and indoor air. *Thorax* 1996; 51: 256-261.

69. Tabbara KF, al Jabarti AL. Hospital construction-associated outbreak of ocular aspergillosis after cataract surgery. *Ophthalmology* 1998; 105: 522-526.

70. Ferre A, Domingo P, Alonso C, Franquet T, Gurgui M, Verger G. Invasive pulmonary aspergillosis: A study of 33 cases. *Med Clin (Barc)* 1998; 110: 421-425. (Spanish)

71. Ewig S, Paar WD, Pakos E et al. Nosocomial ventilator-associated pneumonias caused by *Aspergillus fumigatus* in non-immunosuppressed, non-neutropenic patients. *Pneumologie* 1998; 52: 85-90. (German)

72. Singer S, Singer D, Ruchel R, Mergeryan H, Schmidt U, Harms K. Outbreak of systemic aspergillosis in a neonatal intensive care unit. *Mycoses* 1998; 41: 223-227.

73. Allo MD, Miller J, Townsend T, Tan C. Primary cutaneous aspergillosis associated with Hickman intravenous catheters. *N Engl J Med* 1987; 317: 1105-1108.

74. Boon AP, Adams DH, Buckels J, McMaster P. Cerebral aspergillosis in liver transplantation. *J Clin Pathol* 1990; 43: 114-118.

75. Pla MP, Berenguer J, Arzuaga JA, Banares R, Polo JR, Bouza E. Surgical wound infection by *Aspergillus fumigatus* in liver transplant recipients. *Diagn Microbiol Infect Dis* 1992; 15: 703-706.

76. Kanj SS, Welty-Wolf K, Madden J, et al. Fungal infections in lung and heart-lung transplant recipients. Report of 9 cases and review of the literature. *Medicine (Baltimore)* 1996; 75: 142-156.

77. Pfundstein J. *Aspergillus* infection among solid organ transplant recipients: A case study. *J Transpl Coord* 1997; 7: 187-189.

78. Brenier-Pinchart MP, Lebeau B, Devouassoux G, et al. *Aspergillus* and lung transplant recipients: A mycologic and molecular epidemiologic study. *J Heart Lung Transplant* 1998; 17: 972-979.

79. Gerson SL, Talbot GH, Hurwitz S, Strom B, Lusk EJ. Prolonged granulocytopenia: The major risk factor for invasive pulmonary aspergillosis in patients with leukemia. *Ann Intern Med* 1984; 100: 345-351.

80. Weber SF, Peacock JE Jr, Do KA, Cruz JM, Powell BL, Capizzi RL. Interaction of granulocytopenia and construction activity as risk factors for nosocomial invasive filamentous fungal disease in patients with hematologic disorders. *Infect Control Hosp Epidemiol* 1990; 11: 235-242.

81. Rees JR, Pinner RW, Hajjeh RA, Brandt ME, Reingold AL. The epidemiological features of invasive mycotic

- infections in the San Francisco Bay area, 1992-1993: Results of population-based laboratory active surveillance. *Clin Infect Dis* 1998; 27: 1138-1147.
82. McNeil MM, Nash SL, Hajjeh RA, Conn LA, Plikaytis BD. Trends in mortality due to invasive mycoses in the United States. In: *Program & Abstracts of the International Conference on Emerging Infectious Diseases*. Atlanta, GA, 1998. Abstract S7.3, p. 54.
 83. Wald A, Leisenring W, van Burik JA, Bowden RA. Epidemiology of *Aspergillus* infections in a large cohort of patients undergoing bone marrow transplantation. *J Infect Dis* 1997; 175: 1459-1466.
 84. Gurwith MJ, Stinson EB, Remington JS. *Aspergillus* infection complicating cardiac transplantation: Report of five cases. *Arch Intern Med* 1971; 128:541-545.
 85. Weiland D, Ferguson RM, Peterson PK, Snover DC, Simmons RL, Najarian JS. Aspergillosis in 25 renal transplant patients. *Ann Surg* 1983; 198: 622-629.
 86. Hofflin JM, Potasman I, Baldwin JC, Oyster PE, Stinson EB, Remington JS. Infectious complications in heart transplant recipients receiving cyclosporine and corticosteroids. *Ann Intern Med* 1987; 106: 209-216.
 87. Schulman LL, Smith CR, Drusin R, Rose EA, Enson Y, Reemtsma K. Respiratory complications of cardiac transplantation. *Am J Med Sci* 1988; 296: 1-10.
 88. Gustafson TL, Schaffner W, Lavelly GB, Stratton CW, Johnson HK, Hutcheson RH. Invasive aspergillosis in renal transplant recipients: Correlation with corticosteroid therapy. *J Infect Dis* 1983; 148: 230-238.
 89. Denning DW, Stevens DA. Antifungal and surgical treatment of invasive aspergillosis: Review of 2121 published cases. *Rev Infect Dis* 1990; 12: 1147-1201.
 90. Weinberger M, Elattaar I, Marshall D, et al. Patterns of infection in patients with aplastic anemia and the emergence of *Aspergillus* as a major cause of death. *Medicine* 1992; 71: 24-43.
 91. Noble WC, Clayton YM. Fungi in the air of hospital wards. *J Gen Microbiol* 1963; 32: 397-402.
 92. Solomon WR, Burge HP, Boise JR. Airborne *Aspergillus fumigatus* levels outside and within a large clinical center. *J Allergy Clin Immunol* 1978; 62: 56-60.
 93. Streifel AJ, Rhame FS. Hospital air filamentous fungal spore and particle counts in a specially designed hospital. In: *Indoor Air '93: Proceedings of the Sixth International Conference on Indoor Air and Climate, Vol. 4*. Helsinki, Finland: 161-165.
 94. Rhame FS, Streifel AJ, Kersey JHJ. Extrinsic risk factors for pneumonia in the patient at high risk for infection. *Am J Med* 1984; 76: 42-52.
 95. Rhame FS, Streifel A, Stevens P, et al. Endemic *Aspergillus* airborne spore levels are a major risk factor for aspergillosis in bone marrow transplant patients. Abstracts of the 25th Interscience Conference on Antimicrobial Agents and Chemotherapy 1985. Abstract.
 96. Lentino JR, Rosenkranz MA, Michaels JA, Kurup VP, Rose HD, Rytel MW. Nosocomial aspergillosis: A retrospective review of airborne disease secondary to road construction and contaminated air conditioners. *Am J Epidemiol* 1982; 116: 430-437.
 97. Krasinski K, Holzman RS, Hanna B, Greco MA, Graff M, Bhogal M. Nosocomial fungal infection during hospital renovation. *Infect Control* 1985; 6: 278-282.
 98. Cage AA, Dean DC, Schimert G, Minsley N. *Aspergillus* infection after cardiac surgery. *Arch Surg* 1970; 101: 384-387.
 99. Siegler L, Kennedy MJ. *Aspergillus*, *Fusarium*, and other opportunistic moniliaceous fungi. In: *Manual of Clinical Microbiology*, 7th Ed., Murray PR, Baron EJ, Pfaller MA, Tenover FC, Tenover FC, eds. Washington DC; American Society for Microbiology Press; 1999: p. 1212-1241.
 100. Overberger PA, Wadowsky RM, Schaper MM. Evaluation of airborne particulates and fungi during hospital renovation. *Am Ind Hyg Assoc J* 1995; 56: 706-712.
 101. Breton P, Germaud P, Morin O, Audoin AF, Milpied N, Harousseau JL. Rare pulmonary mycoses in patients with hematologic diseases. *Revue Pneumologie Clinique* 1998; 54: 253-257. (French)
 102. Guarro J, Nucci M, Akiti T, Gené J, Barreiro MDGC, Gonçalves RT. Fungemia due to *Fusarium sacchari* in an immunosuppressed patient. *J Clin Microbiol* 2000; 38: 419-421.
 103. Burton JR, Zachery JB, Bessin R, et al. Diagnosis and effective treatment with amphotericin B. *Ann Intern Med* 1972; 77: 383-388.
 104. Kyriakides GK, Zinneman HHA, Hall WH, et al. Immunologic monitoring and aspergillosis in renal transplant patients. *Am J Surg* 1976; 131: 246-252.
 105. Simmons RB, Price DL, Noble JA, Crow SA, Ahearn DG. Fungal colonization of air filters from hospitals. *Am Ind Hyg Assoc J* 1997; 58: 900-904.

106. Pittet D, Huguenin T, Dharan S, et al. Unusual case of lethal pulmonary aspergillosis in patients with chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 1996; 154 (2 Pt 1): 541-544.
107. Mahoney DH Jr, Steuber CP, Starling KA, et al. An outbreak of aspergillosis in children with acute leukemia. *J Pediatr* 1979; 95: 70-72.
108. Ruutu P, Valtonen V, Tiitonen L, et al. An outbreak of invasive aspergillosis in a hematologic unit. *Scand J Infect Dis* 1987; 19: 347-351.
109. Walsh TJ, Dixon DM. Nosocomial aspergillosis: Environmental microbiology, hospital epidemiology, diagnosis, and treatment. *Eur J Epidemiol* 1989; 5: 131-142.
110. Buffington J, Reporter R, Lasker BA, et al. Investigation of an epidemic of invasive aspergillosis: Utility of molecular typing with the use of random amplified polymorphic DNA probes. *Pediatr Infect Dis J* 1994; 13: 386-393.
111. Gerson SL, Parker P, Jacobs MR, Creger R, Lazarus HM. Aspergillosis due to carpet contamination. *Infect Control Hosp Epidemiol* 1994; 15: 221-223.
112. Fox BC, Chamberlin L, Kulich P, Rae EJ, Webster LR. Heavy contamination of operating room air by *Penicillium* species: Identification of the source and attempts at decontamination. *Am J Infect Control* 1990; 18: 300-306.
113. Chazalet V, Debeauvais J-P, Sarfati J, et al. Molecular typing of environmental and patient isolates of *Aspergillus fumigatus* from various hospital settings. *J Clin Microbiol* 1998; 36: 1494-1500.
114. Loudon KW, Coke AP, Burnie JP, Shaw AJ, Oppenheim BA, Morris CQ. Kitchens as a source of *Aspergillus niger* infection. *J Hosp Infect* 1996; 32: 191-198.
115. Abzug MJ, Gardner S, Glode MP, Cymanski M, Roe MH, Odom LF. Heliport-associated nosocomial mucormycoses. *Infect Control Hosp Epidemiol* 1992; 13: 325-326.
116. Alvarez M, Lopez Ponga B, Rayon C, et al. Nosocomial outbreak caused by *Scedosporium prolificans* (*inflatum*): Four fatal cases in leukemic patients. *J Clin Microbiol* 1995; 33: 3290-3295.
117. Schlepner CJ, Hamilton JR. A pseudoepidemic of pulmonary fungal infections related to fiberoptic bronchoscopy. *Infect Control* 1980; 1: 38-42.
118. Jackson L, Klotz SA, Normand RE. A pseudoepidemic of *Sporothrix cyanescens* pneumonia occurring during renovation of a bronchoscopy suite. *J Med Vet Mycol* 1990; 28: 455-459.
119. Vargo JA, Ginsberg MM, Mizrahi M. Human infestation by the pigeon mite: A case report. *Am J Infect Control* 1983; 11: 24-25.
120. American Institute of Architects. *Guidelines for Design and Construction of Hospital and Health Care Facilities, 2001*. Washington DC; American Institute of Architects Press; 2000: in press.
121. Diamond RD. *Cryptococcus neoformans*. In: *Principles and Practice of Infectious Diseases*, 5th Ed.; Mandell GL, Bennett JE, Dolin R. eds. Philadelphia PA; Churchill Livingstone; 2000: p. 2707-2718.
122. Deepe GS Jr. *Histoplasma capsulatum*. In: *Principles and Practice of Infectious Diseases*, 5th Ed.; Mandell GL, Bennett JE, Dolin R. eds. Philadelphia PA; Churchill Livingstone; 2000: p. 2718-2733.
123. Brodsky AL, Gregg MB, Loewenstein MS, et al. Outbreak of histoplasmosis associated with the 1970 earth day activities. *Am J Med* 1973; 54: 333-342.
124. Ward JI, Weeks M, Allen D, et al. Acute histoplasmosis: Clinical, epidemiologic and serologic findings of an outbreak associated with exposure to a fallen tree. *Am J Med* 1979; 66: 587-595.
125. Galgiani JN. Coccidioidomycoses. In: *Current Clinical Topics in Infectious Disease*; Remington JS, Swartz MN, eds. Malden MA; Blackwell Science; 1997: p. 188-204.
126. Gerberding JL. Nosocomial transmission of opportunistic infections. *Infect Control Hosp Epidemiol* 1998; 19: 574-577.
127. Hughes WT. Natural mode of acquisition for *de novo* infection with *Pneumocystis carinii*. *J Infect Dis* 1982; 145: 842-848.
128. Olsson M, Sukura A, Lindberg LA, et al. Detections of *Pneumocystis carinii* DNA by filtration of air. *Scand J Infect Dis* 1996; 28: 279-282.
129. Bartlett MS, Vermund SH, Jacobs R, et al. Detection of *Pneumocystis carinii* DNA in air samples: Likely environmental risk to susceptible persons. *J Clin Microbiol* 1997; 35: 2511-2513.
130. Lundgren B, Elvin K, Rothman LP, Ljungstrom I, Lidman C, Lundgren JD. Transmission of *Pneumocystis carinii* from patients to hospital staff. *Thorax* 1997; 52: 422-424.
131. Vargas SL, Ponce CA, Gigliotti F, et al. Transmission of *Pneumocystis carinii* DNA from a patient with *P. carinii* pneumonia to immunocompetent contact health care workers. *J Clin Microbiol* 2000; 38: 1536-1538.
132. Walzer PD. *Pneumocystis carinii*. In: *Principles and Practice of Infectious Diseases*, 5th Ed.; Mandell GL, Bennett JE, Dolin R. eds. Philadelphia PA; Churchill Livingstone; 2000: p. 2781-2795.

133. Centers for Disease Control and Prevention. Screening for tuberculosis and tuberculosis infection in high-risk populations: Recommendations of the Advisory Committee for Elimination of Tuberculosis. *MMWR* 1995; 44 (RR-11): 18-34.
134. Centers for Disease Control and Prevention. Targeted tuberculin testing and treatment of latent tuberculosis infection. *MMWR* 2000; 49 (RR-6): 1-51.
135. American Thoracic Society/Centers for Disease Control and Prevention. Diagnostic standards and classification of tuberculosis. *Am Rev Respir Dis* 1990; 142: 725-735.
136. Wells WF. Aerodynamics of droplet nuclei. In: *Airborne Contagion and Air Hygiene*. Cambridge MA; Harvard University Press; 1955: p. 13-19.
137. Haas DW. *Mycobacterium tuberculosis*. In: *Principles and Practice of Infectious Diseases*, 5th Ed.; Mandell GL, Bennett JE, Dolin R. eds. Philadelphia PA; Churchill Livingstone; 2000: p. 2576-2607.
138. Jensen PA. Airborne *Mycobacterium* spp. In: *Manual of Environmental Microbiology*; Hurst CJ, Knudsen GR, McInerney MJ, Stetzenbach LD, Walter MV, eds. Washington DC; American Society for Microbiology Press; 1997: p. 676-681.
139. American Public Health Association. Tuberculosis. In: *Control of Communicable Diseases Manual*, 17th Ed; Chin J, ed. Washington DC; American Public Health Association Press; 2000: p. 521-530.
140. Centers for Disease Control and Prevention. Summary of notifiable diseases, United States 1997. *MMWR* 1998; 46 (54): 1-87.
141. White A. Relation between quantitative nasal cultures and dissemination of staphylococci. *J Lab Clin Med* 1961; 58: 273-277.
142. Huijsmans-Evers AG. Results of routine tests for the detection of dispersers of *Staphylococcus aureus*. *Arch Chir Neerl* 1978; 30: 141-150.
143. Boyce JM, Opal SM, Potter-Bynoe G, Medeiros AA. Spread of methicillin-resistant *Staphylococcus aureus* in a hospital after exposure to a healthcare worker with chronic sinusitis. *Clin Infect Dis* 1993; 17: 496-504.
144. Hambraeus A, Sanderson HF. The control of ventilation of airborne bacterial transfer between hospital patients, and its assessment by means of a particle tracer. 3. Studies with an airborne-particle tracer in an isolation ward for burned patients. *J Hyg (Lond)* 1972; 70: 299-312.
145. Nakashima AK, Allen JR, Martone WJ, et al. Epidemic bullous impetigo in a nursery due to a nasal carrier of *Staphylococcus aureus*: Role of epidemiology and control measures. *Infect Control* 1984; 5: 326-331.
146. Bethune DW, Blowers R, Parker M, Pask EA. Dispersal of *Staphylococcus aureus* by patients and surgical staff. *Lancet* 1965; 1: 480-483.
147. Sherertz RJ, Reagan DR, Hampton KD, et al. A cloud adult: The *Staphylococcus aureus* - virus interaction revisited. *Ann Intern Med* 1996; 124: 539-547.
148. Gryska PF, O'Dea AE. Postoperative streptococcal wound infection. The anatomy of an epidemic. *JAMA* 1970; 213: 1189-1191.
149. Stamm WE, Feeley JC, Facklam RR. Wound infection due to group A *Streptococcus* traced to a vaginal carrier. *J Infect Dis* 1978; 138: 287-292.
150. Berkelman RL, Martin D, Graham DR. Streptococcal wound infection caused by a vaginal carrier. *JAMA* 1982; 247: 2680-2682.
151. McIntyre DM. An epidemic of *Streptococcus pyrogenes* puerpural and postoperative sepsis with an unusual site - the anus. *Am J Obstet Gynecol* 1968; 101: 308-314.
152. Gaynes RP, Horan TC. Surveillance of nosocomial infections. In: *Hospital Epidemiology and Infection Control*, 2nd Ed.; Mayhall CG, ed. Philadelphia PA; Lippincott Williams & Wilkins; 1999: p 1285-1318.
153. Wenzel RP, Veazey JM Jr, Townsend TR. Role of the inanimate environment in hospital-acquired infections. In: *Infection Control in Healthcare Facilities: Microbiological Surveillance*; Cundy KR, Ball W, eds. Baltimore MD: University Park Press; 1977: p. 71-98.
154. Mortimer EA Jr, Wolinsky E, Gonzaga AJ, Rammelkamp CH Jr. Role of airborne transmission in staphylococcal infections. *Br Med J* 1966; 5483: 319-322.
155. Youngs ER, Roberts C, Kramer JM, Gilbert RJ. Dissemination of *Bacillus cereus* in a maternity unit. *J Infect* 1985 10: 228-232.
156. Richardson AJ, Rothburn MM, Roberts C. Pseudo-outbreak of *Bacillus* species: Related to fiberoptic bronchoscopy. *J Hosp Infect* 1986; 7: 208-210.
157. Bryce EA, Smith JA, Tweeddale M, Andruschak BJ, Maxwell MR. Dissemination of *Bacillus cereus* in an intensive care unit. *Infect Control Hosp Epidemiol* 1993; 14: 459-462.

158. Lie PY-F, Ke S-C, Chen S-L. Use of pulsed-field gel electrophoresis to investigate a pseudo-outbreak of *Bacillus cereus* in a pediatric unit. *J Clin Microbiol* 1997; 35: 1533-1535.
159. McDonald LC, Walker M, Carson L, et al. Outbreak of *Acinetobacter* spp. bloodstream infections in a nursery associated with contaminated aerosols and air conditioners. *Pediatr Infect Dis J* 1998; 17: 716-722.
160. Leclair JM, Zaia JA, Levin MJ, Congdon RG, Goldmann DA. Airborne transmission of chickenpox in a hospital. *N Engl J Med* 1980; 302: 450-453.
161. Gustafson TL, Lavelly GB, Brawner ERJ, Hutcheson RHJ, Wright PF, Schaffner W. An outbreak of airborne nosocomial varicella. *Pediatrics* 1982; 70: 550-556.
162. Josephson A, Gombert ME. Airborne transmission of nosocomial varicella from localized zoster. *J Infect Dis* 1988; 158: 238-241.
163. Sawyer MJ, Chamberlin CJ, Wu YN, Aintablian N, Wallace MR. Detection of varicella-zoster virus DNA in air samples from hospital rooms. *J Infect Dis* 1994; 169: 91-94.
164. Menkhaus NA, Lamphear B, Linnemann CC. Airborne transmission of varicella-zoster virus in hospitals. *Lancet* 1990; 226: 1315.
165. Centers for Disease Control and Prevention. Prevention of varicella: Recommendations of the advisory committee on immunization practices (ACIP). *MMWR* 1996; 45 (RR-11): 1-36.
166. Davis RM, Orenstein WA, Frank JAJ, et al. Transmission of measles in medical settings: 1980 through 1984. *JAMA* 1986; 255: 1295-1298.
167. Watkins NM, Smith RPI, St Germain DL, Mackay DN. Measles infection in a hospital setting. *Am J Infect Control* 1987; 15: 201-206.
168. Revera ME, Mason WH, Ross LA, Wright HT Jr. Nosocomial measles infection in a pediatric hospital during a community-wide epidemic. *J Pediatr* 1991; 119: 183-186.
169. Atkinson WL, Markowitz LE, Adams NC, Seastrom GR. Transmission of measles in medical settings - United States, 1985-1989. *Am J Med* 1991; 91: 320S-324S.
170. Patriarca PA, Weber JA, Parker RA, et al. Efficacy of influenza vaccine in nursing homes: Reduction in illness and complications during influenza A (H3N2) epidemics. *JAMA* 1985; 253: 1136-1139.
171. Arden NH, Patriarca PA, Fasano MB, et al. The roles of vaccination and amantadine prophylaxis in controlling an outbreak of influenza A (H3N2) in a nursing home. *Arch Intern Med* 1988; 148: 865-868.
172. Centers for Disease Control and Prevention. Influenza A outbreaks - Louisiana, August 1993. *MMWR* 1993; 42: 132-134.
173. Drinka PJ, Gravenstein S, Krause P, Schilling M, Miller BA, Shult P. Outbreaks of influenza A and B in a highly immunized nursing home population. *J Family Practice* 1997; 45: 509-514.
174. Schilling M, Povinelli L, Krause P, et al. Efficacy of zanamivir for chemoprophylaxis of nursing home influenza outbreaks. *Vaccine* 1998; 16: 1771-1774.
175. Hall CB. Nosocomial viral infections: Perennial weeds on pediatric wards. *Am J Med* 1981; 70: 670-676.
176. Whimbey E, Elting LS, Couch RB, et al. Influenza A virus infections among hospitalized adult bone marrow transplant recipients. *Bone Marrow Transpl* 1994; 13: 437-440.
177. Evans ME, Hall KL, Berry SE. Influenza control in acute care hospitals. *Am J Infect Control* 1997; 25: 357-362.
178. Munoz FM, Campbell JR, Atmar RL, et al. Influenza A virus outbreak in a neonatal intensive care unit. *Pediatr Infect Dis J* 1999; 18: 811-815.
179. Alford RH, Kasel JA, Gerone PJ, Knight V. Human influenza resulting from aerosol inhalation. *Proc Soc Exp Biol Med* 1966; 122: 800-804.
180. Moser MR, Bender TR, Margolis HS, Noble GR, Kendal AP, Ritter DG. An outbreak of influenza aboard a commercial airliner. *Am J Epidemiol* 1979; 110: 1-6.
181. Chanock RW, Kim HW, Vargosko AJ, et al. Respiratory syncytial virus 1. Virus recovery and other observations during 1960. Outbreak of bronchiolitis, pneumonia, and other minor respiratory illness in children. *JAMA* 1961; 176: 647-653.
182. Gardner DS, Court SDM, Brocklebank JT, et al. Virus cross-infection in paediatric wards. *Br Med J* 1973; 2: 571-575.
183. Sawyer LA, Murphy JJ, Kaplan JE, et al. 25-30 nm virus particle associated with a hospital outbreak of acute gastroenteritis with evidence for airborne transmission. *Am J Epidemiol* 1988; 127: 1261-1271.
184. Baxby D. Poxviruses. In: *Textbook of Human Virology*, 2nd Ed.; Belshe RB, ed. St. Louis MO; Mosby Year Book; 1991: p. 930-946.
185. Neff JM. Variola (smallpox) and monkeypox viruses. In: *Principles and Practice of Infectious Diseases*, 5th

Ed.; Mandell GL, Bennett JE, Dolin R. eds. Philadelphia PA; Churchill Livingstone; 2000: p. 1555-1556.

186. Wehrle PF, Posch J, Richter KH, Henderson DA. An airborne outbreak of smallpox in a German hospital and its significance with respect to other recent outbreaks in Europe. *Bull WHO* 1970; 43: 669-679.
187. Hawkes N. Science in Europe: Smallpox death in Britain challenges presumption of laboratory safety. *Science* 1979; 203: 855-856.
188. Eickhoff TC. Airborne nosocomial infection: A contemporary perspective. *Infect Control Hosp Epidemiol* 1994; 15: 663-672.
189. Nuzum EO, Rossi CA, Stephenson EH, LeDuc JW. Aerosol transmission of Hantaan and related viruses to laboratory rats. *Am J Trop Med Hyg* 1988; 38: 636-640.
190. Centers for Disease Control and Prevention. Hantavirus infection - southwestern United States: Interim recommendations for risk reduction. Centers for Disease Control and Prevention. *MMWR* 1993; 42 (RR-11): 1-13.
191. Vitek CR, Breiman RF, Ksiazek TG, et al. Evidence against person-to-person transmission of hantavirus to health care workers. *Clin Infect Dis* 1996; 22: 824-826.
192. Wells RM, Young J, Williams RJ, et al. Hantavirus transmission in the United States. *Emerg Infect Dis* 1997; 3: 361-365.
193. Chaparro J, Vega J, Terry W, et al. Assessment of person-to-person transmission of hantavirus pulmonary syndrome in a Chilean hospital setting. *J Hosp Infect* 1998; 40: 281-285.
194. Nolte KB, Foucar K, Richmond JY. Hantaviral biosafety issues in the autopsy room and laboratory: Concerns and recommendations. *Hum Pathol* 1996; 27: 1253-1254.
195. Stephenson EH, Larson EW, Dominik JW. Effect of environmental factors on aerosol-induced Lassa virus infection. *J Med Virol* 1984; 14: 295-303
196. Monath TP. Lassa fever: Review of epidemiology and epizootiology. *Bull World Health Organ* 1975; 52: 577-592.
197. Monath TP, Casals J. Diagnosis of Lassa fever and the isolation and management of patients. *Bull World Health Organ* 1975; 52: 707-715.
198. Zweighaft RM, Fraser DW, Hattwick MA, et al. Lassa fever: Response to an imported case. *N Engl J Med* 1977; 297: 803-807.
199. Cooper CB, Gransden WR, Webster M, King M, O'Mahony M, Young S, et al. A case of Lassa fever: Experience at St Thomas' hospital. *Br Med J (Clin Res Ed)* 1982; 285: 1003-1005.
200. Centers for Disease Control and Prevention. Update: Management of patients with suspected viral hemorrhagic fever - United States. *MMWR* 1995; 44: 475-479.
201. Decker MD, Schaffner W. Nosocomial diseases of healthcare workers spread by the airborne or contact routes (other than tuberculosis). In: *Hospital Epidemiology and Infection Control*, 2nd Ed.; Mayhall CG, ed. Philadelphia PA; Lippincott Williams & Wilkins; 1999: p. 1101-1126.
202. Fridkin SK, Kremer FB, Bland LA, Padhye A, McNeil MM, Jarvis WR. *Acremonium kiliense* endophthalmitis that occurred after cataract extraction in an ambulatory surgical center and was traced to an environmental reservoir. *Clin Infect Dis* 1996; 22: 222-227.
203. Loeb M, Wilcox L, Thornley D, et al. *Bacillus* species pseudobacteremia following hospital construction. *Can J Infect Control* 1995; 10: 37-40.
204. Olle-Goig JE, Canela-Soler J. An outbreak of *Brucella melitensis* infection by airborne transmission among laboratory workers. *Am J Public Health* 1987; 77: 335-338.
205. Kiel FW, Khan MY. Brucellosis among hospital employees in Saudi Arabia. *Infect Control Hosp Epidemiol* 1993; 14: 268-272.
206. Staszkiwicz J, Lewis CM, Colville J, Zervos M, Band J. Outbreak of *Brucella melitensis* among microbiology laboratory workers in a community hospital. *J Clin Microbiol* 1991; 20: 287-290.
207. Fiori PL, Mastrandrea S, Rappelli P, Cappuccinelli P. *Brucella abortus* infection acquired in microbiology laboratories. *J Clin Microbiol* 2000; 38: 2005-2006.
208. Spinelli JS, Ascher MS, Brooks DL, et al. Q fever crisis in San Francisco: Controlling a sheep zoonosis in a lab animal facility. *Lab Anim* 1981; 10: 29-38.
209. American Conference of Governmental Industrial Hygienists. *Industrial Ventilation: A Manual of Recommended Practice*, 23rd Ed.; Cincinnati OH; American Conference of Governmental Industrial Hygienists, Inc; 1998: p. 1-512.
210. American Society of Heating, Refrigerating and Air-conditioning Engineers, Inc. *Ventilation for Indoor Air Quality*. ASHRAE Standard 62-1999; Atlanta GA; 1999: p. 1-27.

211. Burroughs HEB. Sick building syndrome: Fact, fiction, or facility? In: *A Guide to Managing Indoor Air Quality in Health Care Organizations*; Hansen W., ed. Oakbrook Terrace IL; Joint Commission on Accreditation of Health Care Organizations; 1997: p. 3-13.
212. Robinson TJ, Ouellet AE. Filters and filtration. *ASHRAE J* 1999; April: 65-70.
213. Rutala WA, Jones SM, Worthington JM, Reist PC, Weber DJ. Efficacy of portable filtration units in reducing aerosolized particles in the size range of *Mycobacterium tuberculosis*. *Infect Control Hosp Epidemiol* 1995; 16: 391-398.
214. U.S. Environmental Protection Agency, Office of Air and Radiation and U.S. Department of Health & Human Services, National Institute of Occupational Safety and Health. Building air quality: A guide for building owners and facilities managers. Washington DC; 1991: USEPA EPA/400/1091/033.
215. Rao CY, Burge HA, Chang JCS. Review of quantitative standards and guidelines for fungi in indoor air. *J Air & Waste Manage Assoc* 1996; 46: 899-906.
216. Riley RL, Wells WF, Mills CC, Nyka W, McLean RL. Air hygiene in tuberculosis: quantitative studies of infectivity and control in a pilot ward. *Am Rev Tuberc* 1957; 75: 420-431.
217. Riley RL, Nardell EA. Cleaning the air: The theory and application of UV air disinfection. *Am Rev Respir Dis* 1989; 139: 1286-1294.
218. Riley RL. Ultraviolet air disinfection for control of respiratory contagion. In: *Architectural Design and Indoor Microbial Pollution*, Kundsinn RB, ed; New York NY; Oxford University Press; 1988: p. 174-197.
219. Willmon TL, Hollaender A, Langmuir AD. Studies of the control of acute respiratory diseases among naval recruits. I. A review of a four-year experience with ultraviolet irradiation and dust suppressive measures, 1943 to 1947. *Am J Hyg* 1948; 48: 227-232.
220. Wells WF, Wells MW, Wilder TS. The environmental control of epidemic contagion. I. An epidemiologic study of radiant disinfection of air in day schools. *Am J Hyg* 1942; 35: 97-121.
221. Perkins JE, Bahlke AM, Silverman HF. Effect of ultra-violet irradiation of classrooms on spread of measles in large rural central schools. *Am J Public Health Nations Health* 1947; 37: 529-537.
222. Nagy R. Application and measurement of ultraviolet radiation. *Am Ind Hyg Assoc J* 1964; 25: 274-281.
223. Illuminating Engineering Society. *IES Lighting Handbook*, 4th ed. New York NY; Illuminating Engineering Society; 1966: p. 25-27.
224. Riley RL. Indoor spread of respiratory infection by recirculation of air. *Bull Physiopathol Respir (Nancy)* 1979; 15: 699-705.
225. Menzies D, Pasztor J, Rand T, Bourbeau J. Germicidal ultraviolet irradiation in air conditioning systems: Effect on office worker health and wellbeing: A pilot study. *Occup Environ Med* 1999; 56: 397-402.
226. Riley RL, Permutt S. Room air disinfection by ultraviolet irradiation of upper air. Air mixing and germicidal effectiveness. *Arch Environ Health* 1971; 22: 208-219.
227. Nicas M, Miller SL. A multi-zone model evaluation of the efficacy of upper-room air ultraviolet germicidal irradiation. *Appl Occup Environ Hyg* 1999; 14: 317-328.
228. Kethley TW, Branch K. Ultraviolet lamps for room air disinfection: Effect of sampling location and particle size of bacterial aerosol. *Arch Environ Health* 1972; 25: 205-214.
229. Riley RL, Knight M, Middlebrook G. Ultraviolet susceptibility of BCG and virulent tubercle bacilli. *Am Rev Respir Dis* 1976; 113: 413-418.
230. Collins FM. Relative susceptibility of acid-fast and non-acid-fast bacteria to ultraviolet light. *Appl Microbiol* 1971; 21: 411-413.
231. Riley RL, Permutt S, Kaufman JE. Convection, air mixing, and ultraviolet air disinfection in rooms. *Arch Environ Health* 1971; 22: 200-207.
232. Nardell EA. Fans, filters, or rays? Pros and cons of the current environmental tuberculosis control technologies. *Infect Control Hosp Epidemiol* 1993; 14: 681-685.
233. ECRI. Health devices evaluation of mobile high efficiency filter air cleaners (MHEFACs). 1997; 26: 367-388.
234. American Society of Heating, Refrigerating, and Air-Conditioning Engineers. *1999 ASHRAE Handbook: Heating, Ventilating, and Air-Conditioning Applications*. Chapter 7: Health Care Facilities. Atlanta GA; 1999: p. 7.1-7.13.
235. Elovitz KM. Understanding what humidity does and why. *ASHRAE J* 1999; April: 84-90.
236. Orme I. Patient impact. In: *A Guide to Managing Indoor Air Quality in Health Care Organizations*; Hansen W, ed. Oakbrook Terrace IL; Joint Commission on Accreditation of Healthcare Organizations Publications; 1997: p. 43-52.
237. Gundermann KO. Spread of microorganisms by air-conditioning systems - especially in hospitals. *Ann NY Acad Sci* 1980; 209-217.

238. Anonymous. Roundtable: What engineers want in a ventilation standard. *ASHRAE J* 1998; June: 30-46.
239. U.S. Environmental Protection Agency. Ventilation and Air Quality in Offices. EPA Document #402-F-94-003, Washington, DC; Revision - July 1990.
240. Hermans RD, Streifel AJ. Ventilation design. In: *Workshop on Engineering Controls for Preventing Airborne Infections in Workers in Health Care and Related Facilities*; Bierbaum PJ, Lippman M, eds. Cincinnati OH; NIOSH and CDC; 1993: p. 107-146.
241. Hansen W. The need for an integrated indoor air quality program. In: *A Guide to Managing Indoor Air Quality in Health Care Organizations*; Hansen W, ed. Oakbrook Terrace IL; Joint Commission on Accreditation of Healthcare Organizations Publications; 1997: p. xiii - xviii.
242. Bartley J. Air (HVAC/Laminar Flow). In: *APIC: Infection Control and Applied Epidemiology: Principles and Practice*; Olmsted R, ed. St. Louis MO; Mosby Year Book Publication; 1996: p.103-109.
243. Erickson DS. Personal communication.
244. Levine AS, Siegel SE, Schreiber AD, et al. Protected environments and prophylactic antibiotics. *N Engl J Med* 1973; 288: 477-483.
245. Denning DW, Clemons KV, Hanson LH, Stevens DA. Restriction endonuclease analysis of total cellular DNA of *Aspergillus fumigatus* isolates of geographically and epidemiologically diverse origin. *J Infect Dis* 1990; 162: 1151-1158.
246. Rhame FS. Prevention of nosocomial aspergillosis. *J Hosp Infect* 1991; 18 (Suppl. A): 466-472.
247. Barnes RA, Rogers TR. Control of an outbreak of nosocomial aspergillosis by laminar air-flow isolation. *J Hosp Infect* 1989; 14: 89-94.
248. Roy M-C. The Operating theater: A special environmental area. In: *Prevention and Control of Nosocomial Infections*, 3rd Ed.; Wenzel RP, ed. Baltimore MD; William & Wilkins; 1997: p. 515-538.
249. Pavelchak N, DePersis RP, London M, et al. Identification of factors that disrupt negative air pressurization of respiratory isolation rooms. *Infect Control Hosp Epidemiol* 2000; 21: 191-195.
250. Streifel, AJ. Personal communication.
251. Anderson K. *Pseudomonas pyocyanea* disseminated from an air cooling apparatus. *Med J Austr* 1959; p. 529-532.
252. Shaffer JG, McDade JJ. Airborne *Staphylococcus aureus*. A possible source in air control equipment. *Arch Environ Health* 1963; 5: 547-551.
253. Morey PR. Building-related illness with a focus on fungal issues. In: *A Guide to Managing Indoor Air Quality in Health Care Organizations*; Hansen W, ed. Oakbrook Terrace IL; Joint Commission on Accreditation of Healthcare Organizations Publications; 1997: p. 15-25.
254. Streifel AJ. Recognizing IAQ risk and implementing an IAQ program. In: *A Guide to Managing Indoor Air Quality in Health Care Organizations*; Hansen W, ed. Oakbrook Terrace IL; Joint Commission on Accreditation of Healthcare Organizations Publications; 1997: p. 75-91.
255. Morey PR. Appendix B. Fungal growth checklist. In: *A Guide to Managing Indoor Air Quality in Health Care Organizations*; Hansen W, ed. Oakbrook Terrace IL; Joint Commission on Accreditation of Health Care Organizations Publications; 1997: p. 129-135.
256. U.S. Environmental Protection Agency. Should You Have the Air Ducts in Your Home Cleaned? EPA Document No. 402-K-97-002, Washington, DC; October 1997.
257. Soules WJ. Airflow management techniques. *Clean Rooms* 1993; 2: 18-20.
258. Lawson CN. Commissioning hospitals for compliance. *ASHRAE Trans* 1993; 99 (2).
259. Wadowsky R, Benner S. Distribution of the genus *Aspergillus* in hospital room air conditioners. *Infect Control* 1987; 8: 516-518.
260. Streifel AJ. Aspergillosis and construction. In: *Architectural Design and Indoor Microbial Pollution*; Kundsinn RB, ed. New York NY; Oxford University Press; 1988: p. 198-217.
261. Streifel AJ, Vesley D, Rhame FS. Occurrence of transient high levels of airborne fungal spores. *Proceedings of the 6th Conference on Indoor Air Quality and Climate*; Toronto ON: 1990.
262. Morey R, Williams C. Porous insulation in buildings: A potential source of microorganisms. *Proceedings - Indoor Air '90, 5th International Conference*; Toronto ON; 1990: p. 1-6.
263. Bartley J. Construction. In: *APIC: Infection Control and Applied Epidemiology: Principles and Practice*, Olmsted R, ed. St. Louis MO, Mosby Year Book Publications; 1996: p. 104: 1-6.
264. Harvey MA. Critical-care-unit design and furnishing: Impact on nosocomial infections. *Infect Control Hosp Epidemiol* 1998; 19: 597-601.
265. National Association of Children's Hospitals and Related Institutions. Patient Care Focus Groups 1998.

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266. Bartley JM. APIC State-of-the-art report: The role of infection control during construction in health care facilities. *Am J Infect Control* 2000; 28: 156-169.
267. Carter CD, Barr BA. Infection control issues in construction and renovation. *Infect Control Hosp Epidemiol* 1997; 18: 587-596.
268. Streifel AJ. Maintenance and engineering: Biomedical engineering. In: *APIC Infection Control and Applied Epidemiology: Principles and Practices*, Olmsted R, ed. St. Louis, MO; Mosby Year Book Publications; 1996: p. 111: 1-7.
269. Kennedy V, Barnard B, Hackett B. Use of a risk matrix to determine the level of barrier protection during construction activities. *Hosp Infect Control*, Atlanta, GA; Peachtree Publications; 1997; 2: 27-28.
270. Morey PR. Building-related illness with a focus on fungal issues. In: *A Guide to Managing Indoor Air Quality in Health Care Organizations*, Hansen W, ed. Oakbrook Terrace IL; Joint Commission on Accreditation of Healthcare Organizations Publications; 1997: p. 15-25.
271. Bryce EA, Walker M, Scharf S, et al. An outbreak of cutaneous aspergillosis in a tertiary-care hospital. *Infect Control Hosp Epidemiol* 1996; 17: 170-172.
272. Thio CL, Smith D, Merz WG, et al. Refinements of environmental assessment during an outbreak investigation of invasive aspergillosis in a leukemia and bone marrow transplant unit. *Infect Control Hosp Epidemiol* 2000; 21: 18-23.
273. Kuehn TH, Gacek B, Yang CH, et al. Final report: ASHRAE 804-RP Phase I identification of contaminants, exposures effects, and control options for construction/renovation activities. Atlanta, GA; ASHRAE, Inc. 1995.
274. Kennedy HF, Michie JR, Richardson MD. Air sampling for *Aspergillus* spp. during building activity in a paediatric hospital ward. *J Hosp Infect* 1995; 31: 322-325.
275. Leenders ACAP, van Belkum A, Behrendt M, Luijendijk AD, Verbrugh HA. Density and molecular epidemiology of *Aspergillus* in air and relationship to outbreaks of *Aspergillus* infection. *J Clin Microbiol* 1999; 37: 1752-1757.
276. Rath PM, Ansorg R. Value of environmental sampling and molecular typing of aspergilli to assess nosocomial sources of aspergillosis. *J Hosp Infect* 1997; 37: 47-53.
277. Streifel AJ, Marshall JW. Parameters for ventilation controlled environments in hospitals. In: *Design, Construction, and Operation of Healthy Buildings. IAQ/1997*. Atlanta GA; ASHRAE Press; 1998.
278. Streifel AJ. Air cultures for fungi. In: *Clinical Microbiology Procedures Handbook*; Gilchrist M, sect ed. Washington DC; American Society for Microbiology Press; 1993: p. 11.8.1-11.8.7.
279. American Conference of Governmental Industrial Hygienists (ACGIH): *2000 Threshold Limit Values and Biological Exposure Indices*; Cincinnati OH; American Conference of Governmental Industrial Hygienists, Inc; 2000: p. 1-184.
280. U.S. Department of Labor, Occupational Safety and Health Administration. Air Contaminants Standard. 29 CFR 1910.1000, §1910.1000, Tables Z-1, Z-3. *Fed Reg* 1993; 58: 35338-35351.
281. Leenders A, vanBelkum A, Janssen S, et al. Molecular epidemiology of apparent outbreaks of invasive *Aspergillus* in a hematology ward. *J Clin Microbiol* 1996; 34: 345-351.
282. James MJ, Lasker BA, McNeil MM, Shelton M, Warnock DW, Reiss E. Use of a repetitive DNA probe to type clinical and environmental isolates of *Aspergillus flavus* from a cluster of cutaneous infections in a neonatal intensive care unit. *J Clin Microbiol* 2000; 38: 3612-3618.
283. Skladny H, Buchheidt D, Baust C, et al. Specific detection of *Aspergillus* species in blood and bronchoalveolar lavage samples of immunocompromised patients by two-step PCR. *J Clin Microbiol* 1999; 37: 3865-3871.
284. Symoens F, Bouchara J-P, Heinemann S, Nolard N. Molecular typing of *Aspergillus terreus* isolates by random amplification of polymorphic DNA. *J Hosp Infect* 2000; 44: 273-280.
285. Diaz-Guerra TM, Mellado E, Cuenca-Estrella M, Gaztelurrutia L, Villate Navarro JI, Rodríguez Tudela JL. Genetic similarity among one *Aspergillus flavus* strain isolated from a patient who underwent heart surgery and two environmental strains obtained from the operating room. *J Clin Microbiol* 2000; 38: 2419-2422.
286. Buttner MP, Stetzenbach LD. Monitoring airborne fungal spores in an experimental indoor environment to evaluate sampling methods and the effects of human activity on air sampling. *Appl Environ Microbiol* 1993; 59: 219-226.
287. Sayer WJ, Shean DB, Ghosseiri J. Estimation of airborne fungal flora by the Anderson sampler versus the gravity settling culture plate. *J Allerg* 1969; 44: 214-227.
288. Hay RJ, Clayton YM, Goodley JM. Fungal aerobiology: How, when and where? *J Hosp Infect* 1995; 30 (Suppl):

352-357.

289. Morris G, Kokki MH, Anderson K, Richardson MD. Sampling of *Aspergillus* spores in air. *J Hosp Infect* 2000; 44: 81-92.
290. Iwen PC, Davis JC, Reed EC, Winfield BA, Hinrichs SH. Airborne fungal spore monitoring in a protective environment during hospital construction and correlation with an outbreak of invasive aspergillosis. *Infect Control Hosp Epidemiol* 1994; 15: 303-306.
291. Pegues DA, McNeil MM, Lasker BA, et al. Aspergillosis in a transplant ICU: Possible person-to-person airborne transmission. Abstract 19; Nosocomial Infections/Surgical Infections; In: *Abstracts of the IDSA 37th Annual Meeting*. 1999: p. 25.
292. Goodley JM, Clayton YM, Hay RJ. Environmental sampling for aspergilli during building construction on a hospital site. *J Hosp Infect* 1994; 26: 27-35.
293. American Society of Heating, Refrigerating, and Air-Conditioning Engineers (ASHRAE). The HVAC commissioning process. ASHRAE Guideline 1-1996. Atlanta GA; American Society of Heating, Refrigerating, and Air-Conditioning Engineers Inc.; 1996: 1-48.
294. Mermel LA, Josephson SL, Giorgio CH, Dempsey J, Parenteau S. Association of Legionnaires' disease with construction: Contamination of potable water? *Infect Control Hosp Epidemiol* 1995; 16: 76-81.
295. Loo VG, Bertrand C, Dixon C, et al. Control of construction-associated nosocomial aspergillosis in an antiquated hematology unit. *Infect Control Hosp Epidemiol* 1996; 17: 360-364.
296. Ottney TC. Particle management for HVAC systems. *ASHRAE J* 1993: 26-34.
297. Rautiala S, Reponen T, Nevalainen A, et al. Control of exposure to airborne viable microorganisms during remediation of moldy buildings: Report of three case studies. *Am Ind Hyg Assoc J* 1998; 59: 455-460.
298. Finkelstein LE, Mendelson MH. Infection control challenges during hospital renovation. *Am J Nursing* 1997; 97: 60-61.
299. Opal SM, Asp AA, Cannady PB Jr, Morse PL, Burton LJ, Hammer II PG. Efficacy of infection control measures during a nosocomial outbreak of disseminated aspergillosis associated with hospital construction. *J Infect Dis* 1986; 153: 634-637.
300. Fitzpatrick F, Prout S, Gilleece A, Fenelon LE, Murphy OM. Nosocomial aspergillosis during building work - A multidisciplinary approach. *J Hosp Infect* 1999; 42: 170-171.
301. Garrett DO, Jochimsen E, Jarvis W. Invasive *Aspergillus* spp. infections in rheumatology patients. *J Rheumatol* 1999; 26: 146-149.
302. Hruszkewycz V, Ruben B, Hypes CM, Bostic GD, Staszkiwicz J, Band JD. A cluster of pseudofungemia associated with hospital renovation adjacent to the microbiology laboratory. *Infect Control Hosp Epidemiol* 1992; 13: 147-150.
303. Laurel VL, Meier PA, Astorga A, Dolan D, Brockett R, Rinaldi MG. Pseudoepidemic of *Aspergillus niger* infections traced to specimen contamination in the microbiology laboratory. *J Clin Microbiol* 1999; 37: 1612-1616.
304. Buckner CD, Clift RA, Sanders AJ, et al. Protective environment for marrow transplant recipients. *Ann Intern Med* 1978; 89: 893-901.
305. Murray WA, Streifel AJ, O'Dea TJ, Rhame FS. Ventilation for protection of immune compromised patients. *ASHRAE Trans* 1988; 94: 1185-1191.
306. Streifel AJ, Vesley D, Rhame FS, Murray B. Control of airborne fungal spores in a university hospital. *Environment International* 1989; 12: 441-444.
307. Perry S, Penland WZ. The portable laminar flow isolator: New unit for patient protection in a germ-free environment. In: *Recent Results in Cancer Research*; New York NY; Springer-Verlag; 1970.
308. Rhame FS. Nosocomial aspergillosis: How much protection for which patients? *Infect Control Hosp Epidemiol* 1989; 10: 296-298.
309. Hofflin JM, Potasman I, Baldwin JC, Oyster PE, Stinson EB, Remington JS. Infectious complications in heart transplant recipients receiving cyclosporine and corticosteroids. *Ann Intern Med* 1987; 106: 209-216.
310. Schulman LL, Smith CR, Drusin R, Rose EA, Enson Y, Reemtsma K. Respiratory complications of cardiac transplantation. *Am J Med Sci* 1988; 296: 1-10.
311. Dummer JS, Ho M. Risk factors and approaches to infections in transplant recipients. In: *Principles and Practice of Infectious Diseases*, 5th Ed; Mandell GL, Bennett JE, Dolin R, eds. Philadelphia PA; Churchill Livingstone; 2000: p. 3126-3135.
312. Dummer JS, Ho M. Infections in solid organ transplant recipients. In: *Principles and Practice of Infectious Diseases*, 5th Ed; Mandell GL, Bennett JE, Dolin R, eds. Philadelphia PA; Churchill Livingstone; 2000: p. 3148-3158.

313. Walsh TR, Guttendorf J, Dummer S, et al. The value of protective isolation procedures in cardiac transplant recipients. *Ann Thorac Surg* 1989; 47: 539-545.
314. McNeil M. Personal communication.
315. Yeager CC. Copper and zinc preservatives. In: *Disinfection, Sterilization, and Preservation*, 4th Ed.; Block SS, ed. Philadelphia PA; Lea & Febiger; 1991: p. 358-361.
316. Fennelly KP. Personal respiratory protection against *Mycobacterium tuberculosis*. *Clin Chest Med* 1997; 18: 1-17.
317. Qian Y, Willeke K, Grinshpun SA, Donnelly J. Performance of N95 respirators: Reaerosolization of bacteria and solid particles. *Am Ind Hyg Assoc J* 1997; 58: 876-880.
318. Qian Y, Willeke K, Grinshpun SA, Donnelly J, Coffey CC. Performance of N95 respirators: Filtration efficiency for airborne microbial and inert particles. *Am Ind Hyg Assoc J* 1998; 59: 128-132.
319. Willeke K, Qian Y. Tuberculosis control through respirator wear: Performance of National Institute for Occupational Safety and Health - regulated respirators. *Am J Infect Control* 1998; 26: 139-142.
320. Reponen TA, Wang Z, Willeke K, Grinshpun SA. Survival of mycobacteria on N95 personal respirators. *Infect Control Hosp Epidemiol* 1999; 20: 237-241.
321. Cookson ST, Jarvis WR. Prevention of nosocomial transmission of *Mycobacterium tuberculosis*. *Infect Dis Clin North Am* 1997; 11: 367-409.
322. Centers for Disease Control and Prevention. Nosocomial transmission of multidrug-resistant tuberculosis among HIV-infected persons: Florida and New York, 1988-1991. *MMWR* 1991; 40: 585-591.
323. Centers for Disease Control and Prevention. Outbreak of multidrug-resistant tuberculosis at a hospital - New York City. *MMWR* 1993; 42: 427-434.
324. Beck-Sague CM, Dooley SW, Hutton MD, et al. Hospital outbreak of multidrug-resistant *Mycobacterium tuberculosis*. *JAMA* 1992; 268: 1280-1286.
325. Coronado VG, Beck-Sague CM, Hutton MD, et al. Transmission of multidrug-resistant *Mycobacterium tuberculosis* among persons with human immunodeficiency virus infection in an urban hospital: Epidemiologic and restriction fragment length polymorphism analysis. *J Infect Dis* 1993; 168: 1052-1055.
326. Coronado VG, Valway S, Finelli L, et al. Nosocomial transmission of multidrug-resistant *Mycobacterium tuberculosis* among intravenous drug users with human immunodeficiency virus infection. In: *Abstracts of the Third Annual Meeting of the Society for Hospital Epidemiology of America*, Chicago IL; 1993: Abstract S50.
327. Dooley SW, Villarino ME, Lawrence M, et al. Tuberculosis in a hospital unit for patients infected with the human immunodeficiency virus (HIV): Evidence of nosocomial transmission. *JAMA* 1992; 267: 2632-2634.
328. Edlin BR, Tokars JI, Grieco MH, et al. An outbreak of multidrug-resistant tuberculosis among hospitalized patients with the acquired immunodeficiency syndrome: Epidemiologic studies and restriction fragment length polymorphism analysis. *N Engl J Med* 1992; 326: 1514-1522.
329. Fischl MA, Uttamchandani RB, Daikos GL, et al. An outbreak of tuberculosis caused by multidrug-resistant tubercle bacilli among patients with HIV infection. *Ann Intern Med* 1992; 117: 177-183.
330. Ikeda ARM, Birkhead GS, DeFerdinando Jr GT, et al. Nosocomial tuberculosis: An outbreak of a strain resistant to seven drugs. *Infect Control Hosp Epidemiol* 1995; 16: 152-159.
331. Jarvis WR. Nosocomial transmission of multidrug-resistant *Mycobacterium tuberculosis*. *Res Microbiol* 1992; 144: 117-122.
332. Jarvis WR. Nosocomial transmission of multidrug-resistant *Mycobacterium tuberculosis*. *Am J Infect Control* 1995; 23: 146-151.
333. Jereb JA, Klevens RM, Privett TD, et al. Tuberculosis in health care workers at a hospital with an outbreak of multidrug-resistant *Mycobacterium tuberculosis*. *Arch Intern Med* 1995; 155: 854-859.
334. Moran GJ, McCabe F, Morgan MT, et al. Delayed recognition and infection control for tuberculosis patients in the emergency department. *Ann Emerg Med* 1995; 26: 283-289.
335. Pearson ML, Jereb JA, Frieden TR, et al. Nosocomial transmission of multidrug-resistant *Mycobacterium tuberculosis*: A risk to hospitalized patients and health-care workers. *Ann Intern Med* 1992; 117: 191-196.
336. Tokars JI, Jarvis WR, Edlin BR, et al. Tuberculin skin testing of hospital employees during an outbreak of multidrug-resistant tuberculosis in human immunodeficiency virus (HIV) infected patients. *Infect Control Hosp Epidemiol* 1992; 13: 509-510.
337. Macher JM. The use of germicidal lamps to control tuberculosis in healthcare facilities. *Infect Control Hosp Epidemiol* 1993; 14: 723-729.
338. Hayden CS, Fischbach, M, Johnston OE. A Model for Calculating Air Leakage in Negative Pressure Isolation

Areas. DHHS; NIOSH Report ECTR 212-05c; 1997.

339. DeLuga GF. Differential airflow, pressure, have key relationship in pressurization. *Lab Design* 1997; 2 (5): 6-7.
340. Ayliffe GAJ. Role of the environment of the operating suite in surgical wound infection. *Rev Infect Dis* 1991; 13: S800-S804.
341. Choux M, Genitori L, Lang D, Lena G. Shunt implantation: Reducing the incidence of shunt infection. *J Neurosurg* 1992; 77: 875-880.
342. Edmiston CE Jr, Sinski S, Seabrook GR, Simons D, Goheen MP. Airborne particulates in the OR environment. *AORN J* 1999; 69: 1169-1172.
343. Duhaime AC, Bonner K, McGowan KL, Schut L, Sutton LN, Plotkin S. Distribution of bacteria in the operating room environment and its relation to ventricular shunt infections: A prospective study. *Childs Nerv Syst* 1991; 7: 211-214.
344. Lidwell OM. Clean air at operation and subsequent sepsis in the joint. *Clin Orthop* 1986; 211: 91-102.
345. Nichols RL. The operating room. In: *Hospital Infections*, 3rd ed.; Bennett JV, Brachman PS, eds Boston MA; Little, Brown and Company; 1992: p. 461-473.
346. Laufman H. The operating room. In: *Hospital Infections*, 2nd ed. Bennett JV, Brachman PS, eds. Boston MA/Toronto ON; Little, Brown and Company; 1986: p. 315-323.
347. Pittet D, Ducloux G. Infectious risk factors related to operating rooms. *Infect Control Hosp Epidemiol* 1994; 15: 456-462.
348. Hambræus A. Aerobiology in the operating room - a review. *J Hosp Infect* 1988; 11 (Suppl A): 68-76.
349. Babb JR, Lynam P, Ayliffe GAJ. Risk of airborne transmission in an operating theater containing four ultraclean air units. *J Hosp Infect* 1995; 31: 159-168.
350. Velesco, E, Thuler LCS, Martins CAS, deCastroDias LM, Conalves VMSC. Risk factors for infectious complications after abdominal surgery for malignant disease. *Am J Infect Control* 1996; 24: 1-6.
351. National Academy of Sciences, National Research Council, Division of Medical Sciences, Ad Hoc Committee on Trauma. Postoperative wound infections: the influence of ultraviolet irradiation of the operating room and of various other factors. *Ann Surg* 1964; 160: 1-192.
352. Charnley J. A clean-air operating enclosure. *Br J Surg* 1964; 51: 202-205.
353. Lidwell OM, Lowbury EJJ, Whyte W, Blowers R, Stanley SJ, Lowe D. Effect of ultraclean air in operating rooms on deep sepsis in the joint after total hip or knee replacement: A randomized study. *Br Med J* 1982; 285: 10-14.
354. Hill C, Flamant R, Mazas F, Evrard J. Prophylactic cefazolin versus placebo in total hip replacement: report of a multicentre double-blind randomized trial. *Lancet* 1981; 1: 795-796.
355. Ha'eri GB, Wiley AM. Total hip replacement in a laminar flow environment with special reference to deep infections. *Clin Orthop* 1980; 148: 163-168.
356. Collins DK, Steinhaus K. Total hip replacement without deep infection in a standard operating room. *J Bone Joint Surg* 1976; 58A: 446-450.
357. Taylor GD, Bannister GC, Leeming JP. Wound disinfection with ultraviolet radiation. *J Hosp Infect* 1995; 30: 85-93.
358. U.S. Department of Labor, Occupational Safety and Health Administration. Occupational exposure to tuberculosis; Proposed rule. (29 CFR 1910). *Federal Register* 1997; 62: 544159-54309.
359. Aranha-Creado H, Prince D, Greene K, Brandwein H. Removal of *Mycobacterium* species by breathing circuit filters. *Infect Control Hosp Epidemiol* 1997; 18: 252-254.
360. McCarthy JF. Risk factors for occupational exposures in healthcare professionals. In: *A Guide to Managing Indoor Air Quality in Health Care Organizations*, Hansen W, ed. Oakbrook Terrace IL; Joint Commission on Accreditation of Healthcare Organizations; 1997: p. 27-41.
361. National Institute for Occupational Safety and Health. NIOSH Health Hazard Evaluation and Technical Assistance Report: HETA 85-126-1932; 1988.
362. National Institute for Occupational Safety and Health. NIOSH Health Hazard Evaluation and Technical Assistance Report: HETA 88-101-2008; 1990.
363. National Institute for Occupational Safety and Health. Control of Smoke from Laser/Electric Surgical Procedures. DHHS (NIOSH) Publication 96-128; 1996.
364. Taravella MJ, Weinberg A, Blackburn P, May M. Do intact viral particles survive excimer laser ablation? *Arch Ophthalmol* 1997; 115: 1028-1030.
365. Hagen KB, Kettering JD, Aprecio RM, et al. Lack of virus transmission by the excimer laser plume. *Am J Ophthalmol* 1997; 124: 206-211.

366. Kunachak S, Sithisarn P, Kulapaditharom B. Are laryngeal papilloma virus-infected cells viable in the plume derived from a continuous mode carbon dioxide laser, and are they infectious? A preliminary report on one laser mode. *J Laryng Otol* 1996; 110: 1031-1033.
367. Hughes PSH, Hughes AP. Absence of human papillomavirus DNA in the plume of erbium:YAG laser-treated warts. *J Am Acad Dermatol* 1998; 38: 426-428.
368. Garden JM, O'Bannion K, Sheinitz LS, et al. Papillomavirus in the vapor of carbon dioxide laser treated verrucase. *JAMA* 1988; 125: 1199-1202.
369. Sawchuck WS, Weber JP, Lowry DR, et al. Infectious papillomavirus in the vapour of warts treated with carbon dioxide laser or electrocoagulation: Detection and protection. *J Am Acad Dermatol* 1989; 21: 41-49.
370. Baggish MS, Poiesz BJ, Joret D, et al. Presence of human immunodeficiency virus DNA in laser smoke. *Lasers Surg Med* 1991; 11: 197-203.
371. Capizzi PJ, Clay RP, Battey MJ. Microbiologic activity in laser resurfacing plume and debris. *Lasers Surg Med* 1998; 23: 172-174.
372. McKinley IB Jr, Ludlow MO. Hazards of laser smoke during endodontic therapy. *J Endodont* 1994; 20: 558.
373. Favero MS, Bolyard EA. Microbiologic considerations. Disinfection and sterilization strategies and the potential for airborne transmission of bloodborne pathogens. *Surg Clin North Am* 1995; 75: 1071-1089.
374. Association of Operating Room Nurses. Proposed recommended practices: Laser safety in the practice setting, In: *Standards, Recommended Practices and Guidelines*. AORN J 1999; 267-272.
375. Streifel AJ. Recognizing IAQ risk and implementing an IAQ program. In: *A Guide to Managing Indoor Air Quality in Health Care Organizations*, Hansen W, ed. Oakbrook Terrace IL; Joint Commission on Accreditation of Healthcare Organizations Publishers; 1997: p. 75-91.
376. Kaufman AF, McDade J, Patton C, et al. Pontiac fever: Isolation of the etiologic agent (*Legionella pneumophila*) and demonstration of its mode of transmission. *Am J Epidemiol* 1981; 114: 337-347.
377. Marston BJ, Lipman HB, Breiman RF. Surveillance for Legionnaires' disease: Risk factors for morbidity and mortality related to infection with *Legionella*. *Arch Intern Med* 1994; 154: 2417-2422.
378. Fliermans CD, Cherry WB, Orrison LH, Smith SJ, Tison DL, Pope DH. Ecologic distribution of *Legionella pneumophila*. *Appl Environ Microbiol* 1981; 41: 9-16.
379. Morris GK, Patton CM, Feeley JC, et al. Isolation of the Legionnaires' disease bacterium from environmental samples. *Ann Intern Med* 1979; 90: 664-666.
380. Hsu SC, Martin R, Wentworth BB. Isolation of *Legionella* species from drinking water. *Appl Environ Microbiol* 1984; 48: 830-832.
381. Tison DL, Seidler RJ. *Legionella* incidence and density in potable drinking water. *Appl Environ Microbiol* 1983; 45: 337-339.
382. Parry MF, Stampleman L, Hutchinson JH, et al. Waterborne *Legionella bozemanii* and nosocomial pneumonia in immunosuppressed patients. *Ann Intern Med* 1985; 103: 205-210.
383. England AC, Fraser DW. Sporadic and epidemic nosocomial legionellosis in the United States. Epidemiologic features. *Am J Med* 1981; 70: 707-711.
384. Cohen ML, Broome CV, Paris AL, et al. Fatal nosocomial Legionnaires' disease. Clinical and epidemiological characteristics. *Ann Intern Med* 1979; 90: 611-613.
385. Haley CE, Cohen ML, Halter J, Meyer RD. Nosocomial Legionnaires' disease: A continuing common-source epidemic at Wadsworth Medical Center. *Ann Intern Med* 1979; 90: 583-586.
386. Stout JE, Yu VL, Vickers RM, Shonnard J. Potable water supply as the hospital reservoir for Pittsburgh pneumonia agent. *Lancet* 1982; 1: 471-472.
387. Jimenez P, Torres A, Rodriguez-Roisin R, et al. Incidence and etiology of pneumonia acquired during mechanical ventilation. *Crit Care Med* 1989; 17: 882-885.
388. Arnow PM, Chou T, Weil D, Shapiro EN, Kretzschmar C. Nosocomial Legionnaires' disease caused by aerosolized tap water from respiratory devices. *J Infect Dis* 1982; 146: 460-467.
389. Mastro TD, Fields BS, Breiman RF, Campbell J, Plikaytis BD, Spika JS. Nosocomial Legionnaires' disease and use of medication nebulizers. *J Infect Dis* 1991; 163: 667-670.
390. Dondero TJ Jr, Rendtorff RC, Mallison GF, et al. An outbreak of Legionnaires' disease associated with a contaminated air-conditioning cooling tower. *N Engl J Med* 1980; 302: 365-370.
391. Garbe PL, Davis BJ, Weisfield JS, et al. Nosocomial Legionnaires' disease: Epidemiologic demonstration of cooling towers as a source. *JAMA* 1985; 254: 521-524.
392. O'Mahony MC, Stanwell-Smith RE, Tillett HE, et al. The Stafford outbreak of Legionnaires' disease. *Epidemiol*

Infect 1990; 104: 361-380.

393. Breiman RF, Fields BS, Sanden G, Volmer L, Meier A, Spika J. An outbreak of Legionnaires' disease associated with shower use: Possible role of amoebae. *JAMA* 1990; 263: 2924-2926.
394. Hanrahan JP, Morse DL, Scharf VB, et al. A community hospital outbreak of legionellosis: Transmission by potable hot water. *Am J Epidemiol* 1987; 125: 639-649.
395. Breiman RF, VanLoock FL, Sion JP, et al. Association of "sink bathing" and Legionnaires' disease [Abstract]. In: Abstracts of the 91st Meeting of the American Society for Microbiology, 1991: L18.
396. Struelens MJ, Maes N, Rost F, et al. Genotypic and phenotypic methods for the investigation of a nosocomial *Legionella pneumophila* outbreak and efficacy of control measures. *J Infect Dis* 1992; 166: 22-30.
397. Farrell ID, Barker JE, Miles EP, Hutchinson JCP. A field study of the survival of *Legionella pneumophila* in a hospital hot-water system. *Epidemiol Infect* 1990; 104: 381-387.
398. Stout JE, Yu VL, Best MG. Ecology of *Legionella pneumophila* within water distribution systems. *Appl Environ Microbiol* 1985; 49: 221-228.
399. Sanden GN, Fields BS, Barbaree JM, et al. Viability of *Legionella pneumophila* in chlorine-free water at elevated temperatures. *Curr Microbiol* 1989; 61-65.
400. Schulze-Röbbecke R, Rodder M, Exner M. Multiplication and killing temperatures of naturally occurring legionellae. *Zbl Bakt Hyg B* 1987; 184: 495-500.
401. Habicht W, Muller HE. Occurrence and parameters of frequency of *Legionella* in warm water systems of hospitals and hotels in Lower Saxony. *Zbl Bakt Hyg B* 1988; 186: 79-88.
402. Ciesielski CA, Blaser MJ, Wang WL. Role of stagnation and obstruction of water flow in isolation of *Legionella pneumophila* from hospital plumbing. *Appl Environ Microbiol* 1984; 48: 984-987.
403. Rowbotham TJ. Preliminary report on the pathogenicity of *Legionella pneumophila* for freshwater and soil amoebae. *J Clin Path* 1980; 33: 179-183.
404. Fields BS, Sanden GN, Barbaree JM, et al. Intracellular multiplication of *Legionella pneumophila* in amoebae isolated from hospital hot water tanks. *Curr Microbiol* 1989; 18: 131-137.
405. Hoge CW, Breiman RF. Advances in the epidemiology and control of *Legionella* infections. *Epidemiol Rev* 1991; 13: 329-340.
406. Breiman RF, Butler JC. Legionnaires' disease: Clinical, epidemiological, and public health perspectives. *Semin Respir Infect* 1998; 13: 84-89.
407. Yu, VL. *Legionella pneumophila* (Legionnaires' disease). In: *Principles and Practice of Infectious Diseases*, 5th Ed.; Mandell GL, Bennett JE, Dolin R, eds. Philadelphia PA; Churchill Livingstone; 2000: p. 2424-2435.
408. Muder RR. Other *Legionella* species. In: *Principles and Practice of Infectious Diseases*, 5th Ed.; Mandell GL, Bennett JE, Dolin R, eds. Philadelphia PA; Churchill Livingstone; 2000: p. 2435-2441.
409. Yu VL. Could aspiration be the major mode of transmission for *Legionella*? *Am J Med* 1993; 95: 13-15.
410. Terranova W, Cohen ML, Fraser DW. Outbreak of Legionnaires' disease diagnosed in 1977. *Lancet* 1978; 2: 122-124.
411. Marrie TJ, Haldane D, MacDonald S, et al. Control of endemic nosocomial Legionnaires' disease by using sterile potable water for high risk patients. *Epidemiol Infect* 1991; 107: 591-605.
412. Nechwatal R, Ehret W, Klatte OJ, et al. Nosocomial outbreak of legionellosis in a rehabilitation center: Demonstration of potable water as a source. *Infection* 1993; 21: 235-240.
413. Hoebe CJP, Cluitmanans JJM, Wagenvoort JHT. Two fatal cases of nosocomial *Legionella pneumophila* pneumonia associated with a contaminated cold water supply. *Eur J Clin Microbiol Infect Dis* 1998; 17: 740-749.
414. Helms CM, Viner JP, Sturm RH, et al. Comparative features of pneumococcal, *Mycoplasma*, and Legionnaires' disease pneumonias. *Ann Intern Med* 1979; 90: 543-547.
415. Yu V, Kroboth FJ, Shonnard J, Brown A, McDearman S, Magnussen M. Legionnaires' disease: New clinical perspectives from a prospective pneumonia study. *Am J Med* 1982; 73: 357-361.
416. Jimenez ML, Aspa J, Padilla B, et al. Fiberoptic bronchoscopic diagnosis of pulmonary disease in 151 HIV-infected patients with pneumonitis. *Eur J Clin Microbiol Infect Dis* 1991; 10: 491-497.
417. Lowry PW, Blankenship RJ, Gridley W, et al. A cluster of *Legionella* sternal wound infections due to postoperative topical exposure to contaminated tap water. *N Engl J Med* 1991; 324: 109-112.
418. Shah A, Check F, Baskin S. Legionnaires' disease and acute renal failure: Case report and review. *Clin Infect Dis* 1992; 14: 204-207.
419. Lowry PW, Tompkins LS. Nosocomial legionellosis: A review of pulmonary and extrapulmonary syndromes. *Am J Infect Control* 1993; 21: 21-27.

420. Schlanger G, Lutwick LI, Kurzman M, et al. Sinusitis caused by *L. pneumophila* in a patient with acquired immune deficiency syndrome. *Am J Med* 1984; 77: 957-960.
421. Tompkins LS, Roessler BJ, Redd SC, et al. *Legionella* prosthetic-valve endocarditis. *N Engl J Med* 1988; 318: 530-535.
422. Bock BV, Kirby BD, Edelstein PH, et al. Legionnaires' disease in renal transplant recipients. *Lancet* 1978; 1: 410-413.
423. Kirby BD, Snyder KM, Meyer RD, et al. Legionnaires' disease. Report of 65 nosocomially acquired cases and review of the literature. *Ann Intern Med* 1980; 59: 188-205.
424. Brady MT. Nosocomial Legionnaires' disease in a children's hospital. *J Pediatr* 1989; 115: 46-50.
425. Horie H, Kawakami H, Minoshima K, et al. Neonatal Legionnaires' disease: Histologic findings in an autopsied neonate. *Acta Pathol Jpn* 1992; 42: 427-31.
426. Roig J, Aguilar X, Ruiz J, et al. Comparative study of *Legionella pneumophila* and other nosocomial pneumonias. *Chest* 1991; 99: 344-350.
427. Redd SC, Schuster DM, Quan J, et al. Legionellosis cardiac transplant recipients: Results of a nationwide survey. *J Infect Dis* 1988; 158: 651-653.
428. Seu P, Winston DJ, Olthoft KM, et al. Legionnaires' disease in liver transplant recipients. *Infect Dis Clin Pract* 1993; 2: 109-113.
429. Chow J, Yu VL. *Legionella*: A major opportunistic pathogen in transplant recipients. *Semin Respir Infect* 1998; 13: 132-139.
430. Kool JL, Fiore AE, Kioski CM, et al. More than ten years of unrecognized nosocomial transmission of Legionnaires' disease among transplant patients. *Infect Control Hosp Epidemiol* 1998; 19: 898-904.
431. LeSaux NM, Sekla L, McLeod J, et al. Epidemic of nosocomial Legionnaires disease in renal transplant recipients: A case-control and environmental study. *Can Med Assoc J* 1989; 140: 1047-1053.
432. Berendt RF, Young HW, Allen RG, Knutsen GL. Dose-response of guinea pigs experimentally infected with aerosols of *Legionella pneumophila*. *J Infect Dis* 1980; 141: 186-192.
433. Marston BJ, Plouffe JF, File TM, et al. Incidence of community-acquired pneumonia requiring hospitalization - Results of a population-based active surveillance study in Ohio. *Arch Intern Med* 1997; 157: 1709-1718.
434. Muder RR, Yu VL, McClure JK, Kroboth FJ, Kominos SD, Lumish RN. Nosocomial Legionnaires' disease uncovered in a prospective pneumonia study: Implications for underdiagnosis. *JAMA* 1983; 318: 3184-3188.
435. Brennen C, Vickers JP, Yu VL, Puntereri A, Yee YC. Discovery of occult *Legionella* pneumonia in a long-stay hospital: Results of prospective serologic survey. *Br Med J* 1987; 295: 306-307.
436. Marrie TJ, MacDonald S, Clarke K, Haldane D. Nosocomial Legionnaires' disease: Lessons from a four-year prospective study. *Am J Infect Control* 1991; 19: 79-85.
437. Stout JE, Yu, VL. Current concepts: Legionellosis. *N Engl J Med* 1997; 337: 682-687.
438. Vergis EN, Yu VL. Macrolides are ideal for empiric therapy of community-acquired pneumonia in the immunocompromised host. *Semin Respir Infect* 1998; 13: 322-328.
439. Sopena N, Sabria-Leal M, Pedro-Botet ML, et al. Comparative study of the clinical presentation of *Legionella* pneumonia and other community-acquired pneumonias. *Chest* 1998; 113: 1195-1200.
440. Hirani NA, MacFarlane JT. Impact of management guidelines on the outcome of severe community acquired pneumonia. *Thorax* 1997; 52: 17-21.
441. Lieberman D, Porath A, Schlaeffer F, Boldur L. *L. pneumophila* species community-acquired pneumonia: A review of 56 hospitalized patients. *Chest* 1996; 109: 1243-1249.
442. Ewig S, Bauer T, Hasper E, et al. Value of routine microbial investigation in community-acquired pneumonia treated in a tertiary care center. *Respiration* 1996; 63: 164-169.
443. Marrie TJ, Peeling RW, Fine MJ, et al. Ambulatory patients with community-acquired pneumonia: The frequency of atypical agents and clinical course. *Am J Med* 1996; 101: 508-515.
444. Villarino ME, Stevens LE, Schable B, et al. Risk factors for epidemic *Xanthomonas maltophilia* infection/colonization in intensive care unit patients. *Infect Control Hosp Epidemiol* 1992; 13: 201-206.
445. Burdge DR, Nakielna EM, Noble MA. Case-control and vector studies of nosocomial acquisition of *Pseudomonas cepacia* in adult patients with cystic fibrosis. *Infect Control Hosp Epidemiol* 1993; 14: 127-130.
446. Stephenson JR, Heard SR, Richards MA, Tabaqchali S. Gastrointestinal colonization and septicaemia with *Pseudomonas aeruginosa* due to contaminated thymol mouthwash in immunocompromised patients. *J Hosp Infect* 1985; 6: 369-378.
447. Kolmos HJ, Thusen B, Neilsen SV, Lohmann M, Kristoffersen K, Rosdahl VT. Outbreak of infection in a burns

- unit due to *Pseudomonas aeruginosa* originating from contaminated tubing used for irrigating patients. *J Hosp Infect* 1993; 24: 11-21.
448. Vanholder R, Vanhaecke E, Ringoir S. Waterborne *Pseudomonas* septicemia. *ASAIO Trans* 1990; 36: M215-216.
449. Ehni WF, Reller LB, Ellison RT III. Bacteremia in granulocytopenic patients in a tertiary-care general hospital. *Rev Infect Dis* 1991; 13: 613-619.
450. Gallagher PG, Watanakunakorn C. *Pseudomonas* bacteremia in a community teaching hospital, 1980-1984. *Rev Infect Dis* 1989; 11: 846-852.
451. Centers for Disease Control. Nosocomial infection and pseudoinfection from contaminated endoscopes and bronchoscopes – Wisconsin and Missouri. *MMWR* 1991; 40: 675-678.
452. Kerr JR, Moore JE, Curran MD, et al. Investigation of a nosocomial outbreak of *Pseudomonas aeruginosa* pneumonia in an intensive care unit by random amplification of polymorphic DNA assay. *J Hosp Infect* 1995; 30: 125-131.
453. Brewer SC, Wunderink RG, Jones CB, Leeper KV. Ventilator-associated pneumonia due to *Pseudomonas aeruginosa*. *Chest* 1996; 109: 1019-1022.
454. Rello J, Jubert P, Valles J, et al. Evaluation of outcome for intubated patients with pneumonia due to *Pseudomonas aeruginosa*. *Clin Infect Dis* 1996; 23: 973-978.
455. Henderson A, Kelly W, Wright M. Fulminant primary *Pseudomonas aeruginosa* pneumonia and septicemia in previously well adults. *Intensive Care Med* 1992; 18: 430-432.
456. Torres A, Serra-Battles J, Ferrer A, et al. Severe community acquired pneumonia. Epidemiology and prognostic factors. *Am Rev Respir Dis* 1991; 144: 312-318.
457. Pedersen SS, Koch C, Høiby N, Rosendal K. An epidemic spread of multiresistant *Pseudomonas aeruginosa* in a cystic fibrosis center. *J Antimicrob Chemother* 1986; 17: 505-516.
458. Kubesch P, Dörk T, Wulbrand U, et al. Genetic determinants of airways' colonization with *Pseudomonas aeruginosa* in cystic fibrosis. *Lancet* 1993; 341: 189-193.
459. Koch C, Høiby N. Pathogenesis of cystic fibrosis. *Lancet* 1993; 341: 1065-1069.
460. Worlitzsch D, Wolz C, Botzenart K, et al. Molecular epidemiology of *Pseudomonas aeruginosa* – Urinary tract infections in paraplegic patients. *Zentrabl Hyg Umweltmed* 1989; 189: 175-184.
461. Glenister H, Holton J, Teall A. Urinary tract pressure recording equipment as a source for infection. *J Hosp Infect* 1985; 6: 224-226.
462. Ferroni A., Nguyen L, Pron B, Quense G, Brusset MC, Berche P. Outbreak of nosocomial urinary tract infections due to *Pseudomonas aeruginosa* in a paediatric surgical unit associated with tap water contamination. *J Hosp infect* 1998; 39: 301-307.
463. Marrie TJ, Major H, Gurwith M, et al. Prolonged outbreak of nosocomial urinary tract infection with a single strain of *Pseudomonas aeruginosa*. *Can Med Assoc J* 1978; 119: 593-598.
464. Moore B, Forman A. An outbreak of urinary *Pseudomonas aeruginosa* infection acquired during urological operations. *Lancet* 1966; 2: 929-931.
465. Anderson RJ, Schafer LA, Olin DB, Eickhoff TC. Septicemia in renal transplant recipients. *Arch Surg* 1973; 106: 692-694.
466. Fang G, Brennen C, Wagener M, et al. Use of ciprofloxacin versus use of aminoglycosides for therapy of complicated urinary tract infection: Prospective, randomized clinical and pharmacokinetic study. *Antimicrob Agents Chemother* 1991; 35: 1849-1855.
467. Dorff GJ, Beimer NF, Rosenthal DR, Rytel MW. *Pseudomonas* septicemia: Illustrated evolution of its skin lesions. *Arch Intern Med* 1971; 128: 591-595.
468. Teplitz C. Pathogenesis of *Pseudomonas* vaculitis and septic lesions. *Arch Pathol* 1965; 80: 297-307.
469. Roberts R, Tarpay MM, Marks MI, Nitschke R. Erysipelas-like lesions and hyperesthesia as manifestations of *Pseudomonas aeruginosa* sepsis. *JAMA* 1982; 248: 2156-2157.
470. Duncan BW, Adzick NS, deLorimier AA, et al. Necrotizing fasciitis in childhood. *J Pediatr Surg* 1992; 27: 668-671.
471. McManus AT, Mason AD Jr, McManus WF, Pruitt BA Jr. Twenty-five year review of *Pseudomonas aeruginosa* bacteremia in a burn center. *Eur J Clin Microbiol* 1985; 4: 219-223.
472. Tredget EE, Shankowsky HA, Joffe AM, et al. Epidemiology of infections with *Pseudomonas aeruginosa* in burn patients: The role of hydrotherapy. *Clin Infect Dis* 1992; 15: 941-949.
473. Schlech WF III, Simosen N, Sumarah R, Martin RS. Nosocomial outbreak of *Pseudomonas aeruginosa*

- folliculitis associated with a physiotherapy pool. *Can Med Assoc J* 1986; 134: 909-913.
474. Fang G, Keys TF, Gentry LO, et al. Prosthetic valve endocarditis resulting from nosocomial bacteremia: A prospective, multicenter study. *Ann Intern Med* 1993; 119: 560-567.
475. Cohen PS, Maguire JH, Weinstein L. Infective endocarditis caused by gram-negative bacteria: A review of the literature, 1945-1977. *Prog Cardiovasc Dis* 1980; 22: 205-242.
476. Wise BL, Mathis JL, Jawetz E. Infections of the central nervous system due to *Pseudomonas aeruginosa*. *J Neurosurg* 1969; 31: 432-434.
477. Bray DA, Calcaterra TC. *Pseudomonas* meningitis complicating head and neck surgery. *Laryngoscope* 1976; 86: 1386-1390.
478. Schein OD, Wasson PJ, Boruchoff SA, Kenyon KR. Microbial keratitis associated with contaminated ocular medications. *Am J Ophthalmol* 1988; 105: 361-365.
479. Procope JA. Delayed-onset *Pseudomonas* keratitis after radial keratotomy. *J Cataract Refract Surg* 1997; 23: 1271-1272.
480. Sapico FL, Montgomerie JZ. Vertebral osteomyelitis in intravenous drug abusers: Report of three cases and review of the literature. *Rev Infect Dis* 1980; 2: 196-206.
481. Tindel JR, Crowder JG. Septic arthritis due to *Pseudomonas aeruginosa*. *JAMA* 1971; 218: 559-561.
482. Martone WJ, Tablan OC, Jarvis WR. The epidemiology of nosocomial epidemic *Pseudomonas cepacia* infections. *Eur J Epidemiol* 1987; 3: 222-232.
483. Goldmann DA, Klinger JD. *Pseudomonas cepacia*: Biology, mechanisms of virulence, epidemiology. *J Pediatr* 1986; 108: 806-812.
484. Widmer AF, Wenzel RP, Trilla A, et al. Outbreak of *Pseudomonas aeruginosa* infections in a surgical intensive care unit: Probable transmission via hands of a health care worker. *Clin Infect Dis* 1993; 16: 372-376.
485. Döring G, Hörz M, Ortelt J, et al. Molecular epidemiology of *Pseudomonas aeruginosa* in an intensive care unit. *Epidemiol Infect* 1993; 110: 427-436.
486. Hollyoak V, Allison D, Summers J. *Pseudomonas aeruginosa* wound infection associated with a nursing home whirlpool bath. *CDR Review* 1995; 5: R100-102.
487. Grundmann H, Kropec A, Hartung D, Berner R, Daschner F. *Pseudomonas aeruginosa* in a neonatal intensive care unit: Reservoirs and ecology of the nosocomial pathogen. *J Infect Dis* 1993; 168: 943-947.
488. Martino P, Venditti M, Papa G, Orefici G, Serra P. Water supply as a source of *Pseudomonas aeruginosa* in a hospital for hematological malignancies. *Bollettino dell Istituto Sieroterapico Milanese* 1985; 64: 109-114.
489. Ayliffe GAJ, Babb JR, Collins BJ, Lowbury EJ, Newsom SWB. *Pseudomonas aeruginosa* in hospital sinks. *Lancet* 1974; 2 (7887): 578-81.
490. Kluyver AJ. *Pseudomonas aureofaciens* nov. spec and its pigments. *J Bacteriol* 1956; 72: 406-411.
491. Bosshammer J, Fiedler B, Gudowius P, von der Hardt H, Romling U, Tummeler B. Comparative hygienic surveillance of contamination with pseudomonads in a cystic fibrosis ward over a 4-year period. *J Hosp Infect* 1995; 31: 261-274.
492. Jones F, Bartlett CL. Infections associated with whirlpools and spas. *Soc Appl Bacteriol Symp Series* 1985; 14: 61S-66S.
493. Casewell MW, Slater NG, Cooper JE. Operating theater water-baths as a cause of *Pseudomonas* septicemia. *J Hosp Infect* 1981; 2: 237-247.
494. Rechsteiner J, Landheer JE, de Jong J, van Kregten E, Lindner JG. [Kidney lithotripter as a possible source of hospital infection]. *Nederlands Tijdschrift voor Geneeskunde* 1988; 132: 1849-1859. (Dutch)
495. Taplin D, Mertz PM. Flower vases in hospitals as reservoirs for pathogens. *Lancet* 1973; 2 (7841): 1279-1281.
496. Kaiser AB. Humidifiers and *Pseudomonas* infections. *N Engl J Med* 1970; 283: 708.
497. Levin MH, Olson B, Nathan C, et al. *Pseudomonas* in the sinks in an intensive care unit: Relation to patients. *J Clin Pathol* 1984; 37: 424-427.
498. Paszko-Kolva C, Yamamoto H, Shahamat M, Sawyer TK, Morris G, Colwell RR. Isolation of amoebae and *Pseudomonas* and *Legionella* spp. from eyewash stations. *Appl Environ Microbiol* 1991; 57: 163-167.
499. Boukadida J, de Montalembert M, Gaillard JL, et al. Outbreak of gut colonization by *Pseudomonas aeruginosa* in immunocompromised children undergoing total digestive decontamination: Analysis by pulsed-field electrophoresis. *J Clin Microbiol* 1991; 29: 2068-2071.
500. Grigis A, Goglio A, Parea M, Gneccchi F, Minetti B, Barbui T. Nosocomial outbreak of severe *Pseudomonas aeruginosa* infections in haematological patients. *Eur J Epidemiol* 1993; 9: 390-395.
501. Gupta AK, Shashi S, Mohan M, Lamba IM, Gupta R. Epidemiology of *Pseudomonas aeruginosa* infections in a

- neonatal intensive care unit. *J Trop Pediatr* 1993; 39: 32-36.
502. Sader HS Pignatari AC, Leme IL, et al. Epidemiologic typing of multiply drug-resistant *Pseudomonas aeruginosa* isolated from an outbreak in an intensive care unit. *Diagn Microbiol Infect Dis* 1993; 17: 13-18.
503. Krecmery V, Trupl J. Nosocomial outbreak of meropenem-resistant *Pseudomonas aeruginosa* infections in a cancer center. *J Hosp Infect* 1994; 28: 209-218.
504. Jumaa P, Chattopadhyay B. Outbreak of gentamicin, ciprofloxacin-resistant *Pseudomonas aeruginosa* in an intensive care unit, traced to contaminated quivers. *J Hosp Infect* 1994; 28: 209-218.
505. Carson LA, Favero MS, Bond WW, Petersen NJ. Morphological, biochemical, and growth characteristics of *Pseudomonas cepacia* from distilled water. *Appl Microbiol* 1973; 25: 476-483.
506. Bassett DC, Stokes KJ, Thomas WR. Wound infection with *Pseudomonas multivorans*: A waterborne contaminant of disinfectant solutions. *Lancet* 1970; 1: 1188-1191.
507. Wishart MM, Riley TV. Infection with *Pseudomonas maltophilia*: Hospital outbreak due to contaminated disinfectant. *Med J Aust* 1976; 2: 710-712.
508. Conly JM, Klass L, Larson L. *Pseudomonas cepacia* colonization and infection in intensive care units. *Can Med Assoc J* 1986; 134: 363-366.
509. Bosshammer J, Fielder B, Gudowis P, et al. Comparative hygienic surveillance of contamination with pseudomonads in a cystic fibrosis ward over a 4-year period. *J Hosp Infect* 1995; 31: 261-274.
510. Hutchinson GR, Parker S, Pryor JA, et al. Home-use nebulizers: A potential primary source of *B. cepacia* and other colistin-resistant, gram-negative bacteria in patients with cystic fibrosis. *J Clin Microbiol* 1996; 34: 584-587.
511. Pegues DA, Carson LA, Anderson RL, et al. Outbreak of *Pseudomonas cepacia* bacteremia in oncology patients. *Clin Infect Dis* 1993; 16: 407-411.
512. Centers for Disease Control and Prevention. Nosocomial *Burkholderia cepacia* infection and colonization with intrinsically contaminated mouthwash - Arizona, 1998. *MMWR* 1998; 47: 926-928.
513. Berthelot P, Grattard F, Mahul P, et al. Ventilator temperature sensors: An unusual source of *Pseudomonas cepacia* in nosocomial infection. *J Hosp Infect* 1993; 25: 33-43.
514. Khardori N, Elting L, Wong E, et al. Nosocomial infections due to *Xanthomonas maltophilia* (*Pseudomonas maltophilia*) in patients with cancer. *Rev Infect Dis* 1990; 12: 997-1003.
515. Oie S, Oomaki M, Yorioka K, et al. Microbial contamination of "sterile water" used in Japanese hospitals. *J Hosp Infect* 1998; 38: 61-65.
516. Crane LR, Tagle LC, Palutke WA. Outbreak of *Pseudomonas paucimobilis* in an intensive care facility. *JAMA* 1981; 246: 985-987.
517. Lemaitre D, Elaichouni A, Hundhausen M, et al. Tracheal colonization with *Sphingomonas paucimobilis* in mechanically-ventilated neonates due to contaminated ventilator temperature probes. *J Hosp Infect* 1996; 32: 199-206.
518. Maki DG, Klein BS, McCormick RD, et al. Nosocomial *Pseudomonas pickettii* bacteremias traced to narcotic tampering. A case for selective drug screening of health care personnel. *JAMA* 1991; 265: 981-986.
519. Maroye P, Doermann HP, Rogues AM, Gachie JP, Mégraud F. Investigation of an outbreak of *Ralstonia pickettii* in a paediatric hospital by RAPD. *J Hosp Infection* 2000; 44: 267-272.
520. McNeil MM, Solomon SL, Anderson RL, et al. Nosocomial *Pseudomonas pickettii* colonization associated with a contaminated respiratory therapy solution in a special care nursery. *J Clin Microbiol* 1985; 22: 903-907.
521. Lamka KG, LeChevallier MW, Seidler RJ. Bacterial contamination of drinking water supplies in a modern rural neighborhood. *Appl Environ Microbiol* 1980; 39: 734-738.
522. Nakashima AK, McCarthy MA, Martone WJ, Anderson RL. Epidemic septic arthritis caused by *Serratia marcescens* and associated with a benzalkonium chloride antiseptic. *J Clin Microbiol* 1987; 25: 1014-1018.
523. Nakashima AK, Highsmith AK, Martone WJ. Survival of *Serratia marcescens* in benzalkonium chloride and in multiple-dose medication vials: Relationship to epidemic septic arthritis. *J Clin Microbiol* 1987; 25: 1019-1021.
524. Bosi C, Davin-Regli A, Charrel R, Rocca B, Monnet D, Bollet C. *Serratia marcescens* nosocomial outbreak due to contamination of hexetidine solution. *J Hosp Infect* 1996; 33: 217-224.
525. Ehrenkranz NJ, Bolyard EA, Wiener M, Cleary TJ. Antibiotic-sensitive *Serratia marcescens* infections complicating cardiopulmonary operations: Contaminated disinfectant as a reservoir. *Lancet* 1980; 2: 1289-1292.
526. Cimolai N, Trombley C, Wensley D, LeBlanc J. Heterogeneous *Serratia marcescens* genotypes from a nosocomial pediatric outbreak. *Chest* 1997; 111: 194-197.
527. Hartstein AI, Rashad AL, Liebler JM, et al. Multiple intensive care unit outbreaks of *Acinetobacter calcoaceticus* subspecies *anitratus* respiratory infection and colonization associated with contaminated, reusable ventilator circuits and resuscitation bags. *Am J Med* 1988; 85: 624-631.

528. Stone JW, Das BC. Investigation of an outbreak of infection with *Acinetobacter calcoaceticus* in a special care baby unit. *J Hosp Infect* 1986; 7: 42-48.
529. Vandenbroucke-Grauls CMJE, Kerver AJH, Rommes JH, Jansen R, den Dekker C, Verhoef J. Endemic *Acinetobacter anitratus* in a surgical intensive care unit: Mechanical ventilators as reservoir. *Eur J Clin Microbiol Infect Dis* 1988; 7: 485-489.
530. Cefai C, Richards J, Gould FK, McPeake P. An outbreak of *Acinetobacter* respiratory infection resulting from incomplete disinfection of ventilatory equipment. *J Hosp Infect* 1990; 15: 177-182.
531. Gervich DH, Grout CS. An outbreak of nosocomial *Acinetobacter* infections from humidifiers. *Am J Infect Control* 1985; 13: 210-215.
532. Castle M, Tenney JH, Weinstein MP, Eickhoff TC. Outbreak of a multiply resistant *Acinetobacter* in a surgical intensive care unit. *Heart Lung* 1978; 7: 641-644.
533. Smith PW, Massanari RM. Room humidifiers as a source of *Acinetobacter* infections. *JAMA* 1977; 237: 795-797.
534. Snyderman DR, Maloy MF, Brock SM, Lyons RW, Rubin SJ. Pseudobacteremia: False-positive blood cultures from mist tent contamination. *Am J Epidemiol* 1977; 106: 154-159.
535. Rosenthal SL. Sources of *Pseudomonas* and *Acinetobacter* species found in human culture materials. *Am J Clin Pathol* 1974; 62: 807-811.
536. Allen KD, Green HT. Hospital outbreak of multi-resistant *Acinetobacter anitratus*: An airborne mode of spread. *J Hosp Infect* 1987; 9: 169-175.
537. Crombach WHJ, Dijkshoorn L, van Noort-Klaassen M, Niessen J, van Knippenbert-Gordebeke G. Control of an epidemic spread of multi-resistant *Acinetobacter calcoaceticus* in a hospital. *Intensive Care Med* 1989; 15: 166-170.
538. Catalano M, Quelle LS, Jeric PE, Di Martino A, Maimone SM. Survival of *Acinetobacter baumannii* on bed rails during an outbreak and during sporadic cases. *J Hosp Infect* 1999; 42: 27-35.
539. D'Agata EMC, Venkataraman L, DeGirolami P, Samore M. Molecular epidemiology of ceftazidime-resistant gram-negative bacilli on inanimate surfaces and their role in cross-transmission during non-outbreak periods. *J Clin Microbiol* 1999; 37: 3065-3067.
540. Jawad A, Snelling AM, Heritage J, Hawkey PM. Exceptional desiccation tolerance of *Acinetobacter radioresistens*. *J Hosp Infect* 1998; 39: 235-240.
541. Jawad A, Seifert H, Snelling AM, Heritage J, Hawkey PM. Survival of *Acinetobacter baumannii* on dry surfaces: Comparison of outbreak and sporadic isolates. *J Clin Microbiol* 1998; 36: 1938-1941.
542. Getschell-White, SI, Donowitz LG, Groschel DHM. The inanimate environment of an intensive care unit as a potential source of nosocomial bacteria: Evidence for long survival of *Acinetobacter calcoaceticus*. *Infect Control Hosp Epidemiol* 1989; 10: 402-406.
543. Loiwal V, Kumar A, Gupta P, Gomber S, Ramachandran VG. *Enterobacter aerogenes* outbreak in a neonatal intensive care unit. *Pediatr Int* 1999; 41: 157-161.
544. Matsaniotis NS, Syriopoulou VP, Theodoridou MC, Tzanetou KG, Mostrou GI. *Enterobacter* sepsis in infants and children due to contaminated intravenous fluids. *Infect Control* 1984; 5: 471-477.
545. Zembrzuska-Sadlowska E. The dangers of infections of the hospitalized patients with the microorganisms present in preparations and in the hospital environment. *Acta Pol Pharm* 1995; 52: 173-178.
546. Felts SK, Schaffner W, Melly MA, Koenig MG. Sepsis caused by contaminated intravenous fluids. *Ann Intern Med* 1972; 77: 881-890.
547. Modi N, Damjanovic V, Cooke RW. Outbreak of cephalosporin resistant *Enterobacter cloacae* infection in a neonatal intensive care unit. *Arch Dis Child* 1987; 62: 148-151.
548. Graham DR, Wu E, Highsmith AK, Ginsburg ML. An outbreak of pseudobacteremia caused by *Enterobacter cloacae* from a phlebotomist's vial of thrombin. *Ann Intern Med* 1981; 95: 585-588.
549. Andersen BM, Sorlie D, Hotvedt R, et al. Multiply beta-lactam-resistant *Enterobacter cloacae* infections linked to the environmental flora in a unit for cardiothoracic and vascular surgery. *Scand J Infect Dis* 1989; 21: 181-191.
550. Wisplinghoff H, Perbix W, Seifert H. Risk factors for nosocomial bloodstream infections due to *Acinetobacter baumannii*: A case-control study of adult burn patients. *Clin Infect Dis* 1999; 28: 59-66.
551. Crowe M, Towner KJ, Humphreys H. Clinical and epidemiological features of an outbreak of *Acinetobacter* infection in an intensive therapy unit. *J Med Microbiol* 1995; 43: 55-62.
552. National Nosocomial Infections Surveillance (NNIS) Report: Data summary from October 1986-April 1996, issued May 1996. *Am J Infect Control* 1996; 24: 380-388.
553. Bergogne-Bérézin E, Joly-Guillou ML. Hospital infection with *Acinetobacter* spp.: An increasing problem. *J Hosp Infect* 1991; 18 (Suppl A): 250-255.

554. Fagon JY, Chastre J, Hance AJ, Montravers P, Novara A, Gibert C. Nosocomial pneumonia in ventilated patients: A cohort study evaluating attributable mortality and hospital stay. *Am J Med* 1993; 94: 281-288.
555. Seifert H, Strate A, Pulverer G. Nosocomial bacteremia due to *Acinetobacter baumannii*: Clinical features, epidemiology, and predictors of mortality. *Medicine* 1995; 74: 340-349.
556. Cisneros JM, Reyes MJ, Pachón J, et al. Bacteremia due to *Acinetobacter baumannii*: Epidemiology, clinical findings, and prognostic features. *Clin Infect Dis* 1996; 22: 1026-1032.
557. Schaberg DR, Culver DH, Gaynes RP. Major trends in the microbial ecology of nosocomial infections. *Am J Med* 1991; 91 (Suppl 3B): 72S-75S.
558. Wang CC, Chu ML, Ho LJ, Hwang RC. Analysis of plasmid pattern in pediatric intensive care outbreaks of nosocomial infection due to *Enterobacter cloacae*. *J Hosp Infect* 1991; 19: 33-40.
559. Acolet D, Ahmet Z, Houang E, Hurley R, Kaufman ME. *Enterobacter cloacae* in a neonatal intensive care unit: Account of an outbreak and its relationship to use of third generation cephalosporins. *J Hosp Infect* 1994; 28: 273-286.
560. Mayhall CG, Lamb VA, Gayle WE Jr, Haynes BW Jr. *Enterobacter cloacae* septicemia in a burn center: Epidemiology and control of an outbreak. *J Infect Dis* 1979; 139: 166-171.
561. John JF Jr, Sharbaugh RJ, Bannister ER. *Enterobacter cloacae*: Bacteremia, epidemiology, and antibiotic resistance. *Rev Infect Dis* 1982; 4: 13-28.
562. McDonald C, Banerjee SN, Jarvis WR, NNIS. Seasonal variation of *Acinetobacter* infections: 1987-1996. *Clin Infect Dis* 1999; 29: 1133-1137.
563. Beck-Sague CM, Jarvis WR, Brook JH, et al. Epidemic bacteremia due to *Acinetobacter baumannii* in five intensive care units. *Am J Epidemiol* 1990; 132: 723-733.
564. Yu VL. *Serratia marcescens*: Historical perspective and clinical review. *N Engl J Med* 1979; 300: 887-893.
565. Wenger PN, Tokars JL, Brennan P, et al. An outbreak of *Enterobacter hormaechei* infection and colonization in an intensive care nursery. *Clin Infect Dis* 1997; 24:1243-1244.
566. Buxton AE, Anderson RL, Wedegar D, Atlas E. Nosocomial respiratory tract infection and colonization with *Acinetobacter calcoaceticus*. *Am J Med* 1978; 65: 507-513.
567. French GL, Casewell MW, Roncoroni AJ, Knight S, Philipps I. A hospital outbreak of antibiotic-resistant *Acinetobacter anitratus*: Epidemiology and control. *J Hosp Infect* 1980; 1: 125-131.
568. Guenter SH, Hendley JO, Wenzel RP. Gram-negative bacilli as nontransient flora on the hands of hospital personnel. *J Clin Microbiol* 1987; 25: 488-490.
569. Dreyfuss D, Djedaini K, Weber P, et al. Prospective study of nosocomial pneumonia and of patient and circuit colonization during mechanical ventilation with circuit changes every 48 hours versus no change. *Am Rev Respir Dis* 1991; 143: 738-743.
570. Go SE, Urban C, Burns J, et al. Clinical and molecular epidemiology of *Acinetobacter* infections sensitive only to polymixin B and sublactam. *Lancet* 1994; 344: 1329-1332.
571. Musa EK, Desai N, Casewell MW. The survival of *Acinetobacter calcoaceticus* inoculated on fingertips and on formica. *J Hosp Infect* 1990; 15: 219-227.
572. Jawad A, Heritage J, Snelling AM, Gascoyne-Binzi DM, Hawkey PM. Influence of relative humidity and suspending menstrua on survival of *Acinetobacter* spp. on dry surfaces. *J Clin Microbiol* 1996; 34: 2881-2887.
573. Mulin B, Talon D, Viel JF, et al. Risk factors for nosocomial colonization with multiresistant *Acinetobacter baumannii*. *Eur J Clin Microbiol Infect Dis* 1995; 14: 569-576.
574. O'Brien RJ. The epidemiology of nontuberculous mycobacterial disease. *Clin Chest Med* 1989; 10: 407-418.
575. Böttger EC, Teske A, Kirschner P, et al. Disseminated "*Mycobacterium genavense*" infection in patients with AIDS. *Lancet* 1992; 340: 76-80.
576. Wallace RJ Jr, Brown BA, Griffith DE. Nosocomial outbreaks/pseudo-outbreaks caused by nontuberculous mycobacteria. *Ann Rev Microbiol* 1998; 52: 453-490.
577. Chapman JS, Dewlett HJ, Potts WE. Cutaneous reactions to unclassified mycobacterial antigens. A study of children in household contact with patients who excrete unclassified mycobacteria. *Am Rev Respir Dis* 1962; 86: 547-552.
578. Crow HE, Corpe RF, Smith CE. Is serious pulmonary disease caused by non-photochromogenic ("atypical") acid-fast mycobacteria communicable? *Chest* 1961; 39: 372-381.
579. Kuritsky JM, Bullen MG, Broome CV, Silcox VA, Good RC, Wallace, RJ Jr. Sternal wound infections and endocarditis due to organisms of the *Mycobacterium fortuitum* complex. *Ann Intern Med* 1983; 98: 938-939.
580. Laussucq S, Baltch AL, Smith RP, et al. Nosocomial *Mycobacterium fortuitum* colonization from a contaminated ice machine. *Am Rev Respir Dis* 1988; 138: 891-894.

581. Panwalker AP, Fuhse E. Nosocomial *Mycobacterium gordonae* pseudoinfection from contaminated ice machines. *Infect Control* 1986; 7: 67-70.
582. Wallace RJ Jr, Musser JM, Hull SI, et al. Diversity and sources of rapidly growing mycobacteria associated with infections following cardiac surgery. *J Infect Dis* 1989; 159: 708-716.
583. Burns DN, Wallace RJ Jr, Schultz ME, et al. Nosocomial outbreak of respiratory tract colonization with *Mycobacterium fortuitum*: Demonstration of the usefulness of pulsed-field gel electrophoresis in an epidemiologic investigation. *Am Rev Respir Dis* 1991; 144: 1153-1159.
584. Lessing MPA, Walker MM. Fatal pulmonary infection due to *Mycobacterium fortuitum*. *J. Clin Pathol* 1993; 46: 271-272.
585. Chadha R, Grover M, Sharma A, et al. An outbreak of post-surgical wound infections due to *Mycobacterium abscessus*. *Pediatr Surg Int* 1998; 13: 406-410.
586. Von Reyn CF, Maslow JN, Barber TW, Falkinham JO III, Arbeit RD. Persistent colonization of potable water as a source of *Mycobacterium avium* infection in AIDS. *Lancet* 1994; 343: 1137-1141.
587. du Moulin GC, Stottmeier KD, Pelletier PA, Tsang AY, Hedley-Whyte J. Concentration of *Mycobacterium avium* by hospital hot water systems. *JAMA* 1988; 260: 1599-1601.
588. Peters M, Müller C, Rüscher-Gerdes S, et al. Isolation of atypical mycobacteria from tap water in hospitals and homes: Is this a possible source of disseminated MAC infection in AIDS patients? *J Infect* 1995; 31: 39-44.
589. Soto LE, Bobadilla M, Villalobos Y, et al. Post-surgical nasal cellulitis outbreak due to *Mycobacterium chelonae*. *J Hosp Infect* 1991; 19: 99-106.
590. Wenger JD, Spika JS, Smithwick RW, et al. Outbreak of *Mycobacterium chelonae* infection associated with use of jet injectors. *JAMA* 1990; 264: 373-376.
591. Safranek TJ, Jarvis WR, Carson LA, et al. *Mycobacterium chelonae* wound infections after plastic surgery employing contaminated gentian violet skin-marking solution. *N Eng J Med* 1987; 317: 197-201.
592. Gremillion DH, Mursch SB, Lerner CJ. Injection site abscesses caused by *Mycobacterium chelonae*. *Infect Control* 1983; 4: 25-28.
593. Begg N, O'Mahoney M, Penny P, Richardson AE. *Mycobacterium chelonae* associated with a hospital hydrotherapy pool. *Community Med* 1986; 8: 348-350.
594. Aubuchon C, Hill JJ Jr, Graham DR. Atypical mycobacterial infection of soft tissue associated with use of a hot tub. A case report. *J Bone Joint Surg* 1986; 68-A: 766-768.
595. Kirk J, Kaminski GW. *Mycobacterium marinum* infection. *Aust J Dermatol* 1976; 17: 111-116.
596. Ross BC, Johnson PDR, Oppedisano F, et al. Detection of *Mycobacterium ulcerans* in environmental samples during an outbreak of ulcerative disease. *Appl Environ Microbiol* 1997; 63: 4135-4138.
597. Cox R, deBorja K, Bach MC. A pseudo-outbreak of *Mycobacterium chelonae* infections related to bronchoscopy. *Infect Control Hosp Epidemiol* 1997; 18: 136-137.
598. Hoy J, Rolston K, Hopfer RL. Pseudoepidemic of *Mycobacterium fortuitum* in bone marrow cultures. *Am J Infect Control* 1987; 15: 268-271.
599. Stine TM, Harris AA, Levin S, Rivera N, Kaplan, RL. A pseudoepidemic due to atypical mycobacteria in a hospital water supply. *JAMA* 1987; 258: 809-811.
600. Tokars JI, McNeil MM, Tablan OC, et al. *Mycobacterium gordonae* pseudoinfection associated with a contaminated antimicrobial solution. *J Clin Microbiol* 1990; 28: 2765-2769.
601. Wright EP, Collins CH, Yates MD. *Mycobacterium xenopi* and *Mycobacterium kansasii* in a hospital water supply. *J Hosp Infect* 1985; 6: 175-178.
602. Lockwood WW, Friedman C, Bus N, Pierson C, Gaynes R. An outbreak of *Mycobacterium terrae* in clinical specimens associated with a hospital potable water supply. *Am Rev Respir Dis* 1989; 140: 1614-1617.
603. Sniadack DH, Ostroff SM, Karlix MA, et al. A nosocomial pseudo-outbreak of *Mycobacterium xenopi* due to a contaminated water supply: Lessons in prevention. *Infect Control Hosp Epidemiol* 1993; 14: 636-641.
604. Bennett SN, Peterson DE, Johnson DR, Hall WN, Robinson-Dunn B, Dietrich S. Bronchoscopy-associated *Mycobacterium xenopi* pseudoinfections. *Am J Respir Crit Care Med* 1994; 150: 245-250.
605. du Moulin GC, Stottmeier KD. Waterborne mycobacteria: An increasing threat to health. *ASM News* 1986; 10: 525-529.
606. Engel HWB, Berwald LG. The occurrence of *Mycobacterium kansasii* in tapwater. *Tubercle* 1980; 61: 21-26.
607. Kubalek I, Mysak J. The prevalence of environmental mycobacteria in drinking water supply systems in a demarcated region in Czech Republic in the period 1984-1989. *Eur J Epidemiol* 1996; 12: 471-474.
608. Fox C, Smith F, Brogan O, et al. Non-tuberculous mycobacteria in a hospital's piped water supply. *J Hosp*

Infect 1992; 21: 152-154.

609. Aronson T, Holtzman A, Glover N, et al. Comparison of large restriction fragments of *Mycobacterium avium* isolates recovered from AIDS and non-AIDS patients with those of isolates from potable water. *J Clin Microbiol* 1999; 37: 1008-1012.
610. Carson LA, Bland LA, Cusick LB, et al. Prevalence of nontuberculous mycobacteria in water supplies of hemodialysis centers. *Appl Environ Microbiol* 1988; 54: 3122-3125.
611. Carson LA, Petersen NJ, Favero MS, Aguero SM. Growth characteristics of atypical mycobacteria in water and their comparative resistance to disinfectants. *Appl Environ Microbiol* 1978; 36: 839-846.
612. Taylor RH, Falkinham III JO, Norton CD, LeChevallier MW. Chlorine, chloramine, chlorine dioxide, and ozone susceptibility of *Mycobacterium avium*. *Appl Environ Microbiol* 2000; 66: 1702-1705.
613. Schulze-Röbbecke R, Fischeder R. Mycobacteria in biofilms. *Zbl Hyg* 1989; 188: 385-390.
614. Schulze-Röbbecke R, Feldmann C, Fischeder R, Janning B, Exner M, Wahl G. Dental units: An environmental study of sources of potentially pathogenic mycobacteria. *Tubercle Lung Dis* 1995; 76: 318-323.
615. Meisel JL, Perera DR, Meligro C, Rublin CE. Overwhelming watery diarrhea associated a *Cryptosporidium* in an immunosuppressed patient. *Gastroenterology* 1976; 70: 1156-1160.
616. Nime FA, Page DL, Holscher MA, Yardley JH. Acute enterocolitis in a human being infected with the protozoan *Cryptosporidium*. *Gastroenterology* 1976; 70: 592-598.
617. Goldstein ST, Juranek DD, Ravenholt O, et al. Cryptosporidiosis: An outbreak associated with drinking water despite state-of-the-art treatment. *Ann Intern Med* 1996; 124: 459-468.
618. Rose JB. Enteric waterborne protozoa: Hazard and exposure assessment. In: *Safety of Water Disinfection: Balancing Chemical and Microbial Risks*; Craun GF, ed. Washington DC; ILSI Press; 1993: p. 115-126.
619. Juranek DD, Addiss D, Bartlett ME, et al. Cryptosporidiosis and public health: Workshop report. *Jour AWWA* 1995; 87: 69-80.
620. DuPont HL, Chappell CL, Sterling CR, Okhuysen PC, Rose JB, Jakubowski W. The infectivity of *Cryptosporidium parvum* in healthy volunteers. *N Engl J Med* 1995; 332: 855-859.
621. Chappell CL, Okhuysen PC, Sterling CR, Wang C, Jakubowski W, DuPont HL. Infectivity of *Cryptosporidium parvum* in healthy adults with pre-existing anti- *C. parvum* serum immunoglobulin G. *Am J Trop Med* 1999; 60: 157-164.
622. Meinhardt PL, Casemore DP, Miller KB. Epidemiologic aspects of human cryptosporidiosis and the role of waterborne transmission. *Epidemiol Rev* 1996; 18: 118-136.
623. Rose JB. Occurrence and significance of *Cryptosporidium* in water. *Jour AWWA* 1988; 80: 53-58.
624. Rose JB, Gerba CP, Jakubowski W. Survey of potable water supplies for *Cryptosporidium* and *Giardia*. *Environ Sci Technol* 1991; 25: 1393-1400.
625. LeChevallier MW, Norton WD, Lee RG. *Giardia* and *Cryptosporidium* spp. in filtered drinking water supplies. *Appl Environ Microbiol* 1991; 57: 2617-2621.
626. Mackenzie WR, Hoxie NJ, Proctor ME, et al. A massive outbreak in Milwaukee of *Cryptosporidium* infection transmitted through the public drinking water supply. *N Engl J Med* 1994; 331: 161-167.
627. Atherton F, Newman CP, Casemore DP. An outbreak of waterborne cryptosporidiosis associated with a public water supply in the UK. *Epidemiol Infect* 1995; 115: 123-131.
628. Hayes EB, Matte TD, O'Brien TR, et al. Large community outbreak of cryptosporidiosis due to contamination of a filtered public water supply. *N Engl J Med* 1989; 320: 1372-1375.
629. Neill MA, Rice SK, Ahmad NV, Flanigan TP. Cryptosporidiosis: An unrecognized cause of diarrhea in elderly hospitalized patients. *Clin Infect Dis* 1996; 22: 168-170.
630. Rutala WA, Weber DJ. Water as a reservoir of nosocomial pathogens. *Infect Control Hosp Epidemiol* 1997; 18: 609-616.
631. Chadwick P. The epidemiological significance of *Pseudomonas aeruginosa* in hospital sinks. *Can J Public Health* 1976; 67: 323-328.
632. Cordes LG, Wiesenthal AM, Gorman GW, et al. Isolation of *Legionella pneumophila* from hospital shower heads. *Ann Intern Med* 1981; 94: 195-197.
633. Bollin GE, Plouffe JF, Para MF, Hackman B. Aerosols containing *Legionella pneumophila* generated by shower heads and hot-water faucets. *Appl Environ Microbiol* 1985; 50: 1128-1131.
634. Weber DJ, Rutala WA, Blanchet CN, Jordan M, Gergen MF. Faucet aerators: A source of patient colonization with *Stenotrophomonas maltophilia*. *Am J Infect Control* 1999; 27: 59-63.
635. Kappstein I, Grundmann H, Hauer T, Niemeyer C. Aerators as a reservoir of *Acinetobacter junii*: An outbreak of

bacteraemia in paediatric oncology patients. *J Hosp Infect* 2000; 44: 27-30.

636. Dennis P JL, Wright AE, Rutter DA, Death JE, Jones BPC. *Legionella pneumophila* in aerosols from shower baths. *J Hyg (Camb)* 1984; 93: 349-353.

637. American Society of Heating, Refrigerating, and Air-Conditioning Engineers. *ASHRAE Guideline 12-2000: Minimizing the Risk of Legionellosis Associated with Building Water Systems*. Atlanta GA; ASHRAE, Inc.; 2000: p. 1-16.

638. Newsom SWB. Microbiology of hospital toilets. *Lancet* 1972; 2: 700-703.

639. Gerba CP, Wallis C, Melnick JL. Microbiological hazards of household toilets: Droplet production and the fate of residual organisms. *Appl Microbiol* 1975; 30: 229-237.

640. Hlady WG, Mullen RC, Mintz CS, Shelton BG, Hopkins RS, Daikos GL. Outbreak of Legionnaires' disease linked to a decorative fountain by molecular epidemiology. *Am J Epidemiol* 1993; 138: 555-562.

641. Rees JC, Allen KD. Holy water - A risk factor for hospital-acquired infection. *J Hosp Infect* 1996; 32: 51-55.

642. Favero MS, Petersen NJ, Boyer KM, Carson LA, Bond WW. Microbial contamination of renal dialysis systems and associated risks. *Trans Am Soc Artif Int Organs* 1974; 20: 175-183.

643. Favero MS, Petersen NJ, Carson LA, Bond WW, Hindman SH. Gram-negative bacteria in hemodialysis systems. *Health Lab Sci* 1975; 12: 321-334.

644. Favero MS, Petersen NJ. Microbiologic guidelines for hemodialysis systems. *Dialysis Transplant* 1997; 6: 34-36.

645. Abrutyn E, Goodhart GL, Roos K, Anderson R, Buxton A. *Acinetobacter calcoaceticus* outbreak associated with peritoneal dialysis. *Am J Epidemiol* 1978; 107: 328-335.

646. Mader JT, Reinartz JA. Peritonitis during peritoneal dialysis - The role of the preheating water bath. *J Chronic Dis* 1978; 31: 635-641.

647. Kosatsky T, Kleeman J. Superficial and systemic illness related to a hot tub. *Am J Med* 1985; 79: 10-12.

648. McGuckin MB, Thorpe RJ, Abrutyn E. Hydrotherapy: An outbreak of *Pseudomonas aeruginosa* wound infections related to Hubbard tank treatments. *Arch Phys Med Rehabil* 1981; 62: 283-285.

649. Koepke GH, Christopher RP. Contamination of whirlpool baths during treatment of infected wounds. *Arch Phys Med Rehabil* 1965; 46: 261-263.

650. Miller JK, LaForest NT, Hedberg M, Chapman V. Surveillance and control of Hubbard tank bacterial contaminants. *Phys Ther* 1972; 50: 1482-1486.

651. Nelson RM, Reed JR, Kenton DM. Microbiological evaluation of decontamination procedures for hydrotherapy tanks. *Phys Ther* 1972; 52: 919-923.

652. Page CF. The whirlpool bath and cross-infection. *Arch Phys Med Rehabil* 1954; 35: 97-98.

653. Newsom SWB. Hospital infection from contaminated ice. *Lancet* 1968; 2: 620-622.

654. Ravn P, Lundgren JD, Kjaeldgaard P, et al. Nosocomial outbreak of cryptosporidiosis in AIDS patients. *Br Med J* 1991; 302: 277-280.

655. Bangsborg JM, Uldum S, Jensen JS, Bruun BG. Nosocomial legionellosis in three heart-lung transplant patients: Case reports and environmental observations. *Eur J Clin Microbiol Infect Dis* 1995; 14: 99-104.

656. Stout JE, Yu VL, Muraca P. Isolation of *Legionella pneumophila* from the cold water of hospital ice machines: Implications for origin and transmission of the organism. *Infect Control* 1985; 6: 141-146.

657. Cross DF, Benchimol A, Dimond EG. The faucet aerator - A source of *Pseudomonas* infection. *N Engl J Med* 1966; 274: 1430-1431.

658. Brown DG, Baublis J. Reservoirs of *Pseudomonas* in an intensive care unit for newborn infants: Mechanisms of control. *J Pediatr* 1977; 90: 453-457.

659. Perryman FA, Flournoy DJ. Prevalence of gentamicin- and amikacin-resistant bacteria in sink drains. *J Clin Microbiol* 1980; 12: 79-83.

660. Doring G, Horz M, Ortel J, Grupp H, Wolz C. Molecular epidemiology of *Pseudomonas aeruginosa* in an intensive care unit. *Epidemiol Infect* 1993; 110: 427-436.

661. Teres D, Schweers P, Bushnell LS, Hedley-Whyte J, Feingold DS. Sources of *Pseudomonas aeruginosa* infection in a respiratory/surgical intensive care unit. *Lancet* 1973; 1: 415-417.

662. Barbeau J, Tanguay R, Faucher E, et al. Multiparametric analysis of waterline contamination in dental units. *Appl Environ Microbiol* 1996; 62: 3954-3959.

663. Atlas RM, Williams JF, Huntington MK. *Legionella* contamination of dental-unit waters. *Appl Environ Microbiol* 1995; 61: 1208-1213.

664. Fayle SA, Pollard MA. Decontamination of dental unit water systems: A review of current recommendations. *Br*

Dent J 1996; 181: 369-372.

665. Pien FD, Bruce AE. Nosocomial *Ewingella americana* bacteremia in an intensive care unit. *Arch Intern Med* 1986; 146: 111-112.
666. Stiles GM, Singh L, Imazaki G, Stiles QR. Thermodilution cardiac output studies as a cause of prosthetic valve bacterial endocarditis. *J Thorac Cardiovasc Surg* 1984; 88: 1035-1037.
667. Tyndall RL, Lyle MM, Ironside KS. The presence of free-living amoebae in portable and stationary eye wash stations. *Am Ind Hyg Assoc J* 1987; 48: 933-934.
668. Bowman EK, Vass AA, Mackowski R, Owen BA, Tyndall RL. Quantitation of free-living amoebae and bacterial populations in eyewash stations relative to flushing frequency. *Am Ind Hyg Assoc J* 1996; 57: 626-633.
669. Siegman-Igra Y, Shalem A, Berger SA, Livio S, Michaeli D. Should potted plants be removed from hospital wards? *J Hosp Infect* 1986; 7: 82-85.
670. Kates SG, McGinley KJ, Larson EL, Leyden JJ. Indigenous multiresistant bacteria from flowers in hospital and nonhospital environments. *Am J Infect Control* 1991; 19: 156-161.
671. Zanetti F, Stampi S, De L, et al. Water characteristics associated with the occurrence of *Legionella pneumophila* in dental units. *Eur J Oral Sci* 2000; 108: 22-28.
672. Peel MM, Calwell JM, Christopher PJ, Harkness JL, Rouch GJ. *Legionella pneumophila* and water temperatures in Australian hospitals. *Aust NZ J Med* 1985; 15: 38-41.
673. Groothuis DG, Veenendaal HR, Dijkstra HL. Influence of temperature on the number of *Legionella pneumophila* in hot water systems. *J Appl Bacteriol* 1985; 59: 529-536.
674. Plouffe JF, Webster LR, Hackman B. Relationship between colonization of a hospital building with *Legionella pneumophila* and hot water temperatures. *Appl Environ Microbiol* 1983; 46: 769-770.
675. Alary Ma, Joly JR. Factors contributing to the contamination of hospital water distribution systems by *Legionellae*. *J Infect Dis* 1992; 165: 565-569.
676. U.K. Health & Safety Executive. The control of legionellosis in hot and cold water systems. Supplement to: *The Control of Legionellosis, Including Legionnaires' Disease*. Health & Safety Executive Office; London UK; 1998: p. 1-4.
677. Marrie TJ, Haldane D, Bezanson G, Peppard R. Each water outlet is a unique ecologic niche for *Legionella pneumophila*. *Epidemiol Infect* 1992; 108: 261-270.
678. Snyder MB, Siwicki M, Wireman J, et al. Reduction of *Legionella pneumophila* through heat flushing followed by continuous supplemental chlorination of hospital hot water. *J Infect Dis* 1990; 162: 127-132.
679. Ezzeddine H, Van Ossel C, Delmee M, Wauters G. *Legionella* spp. in a hospital hot water system: Effect of control measures. *J Hosp Infect* 1989; 13: 121-131.
680. Reichert M. Automatic washers/disinfectors for flexible endoscopes. *Infect Control Hosp Epidemiol* 1991; 12: 497-499.
681. Best M, Yu VL, Stout J, Goetz A, Muder RR, Taylor F. *Legionellaceae* in the hospital water supply: Epidemiologic link with disease and evaluation of a method for control of nosocomial Legionnaires' disease and Pittsburgh pneumonia. *Lancet* 1983; 2: 307-310.
682. Centers for Disease Control and Prevention. Emergency Response Planning and Coordination. <http://www.cdc.gov/nceh/emergency/emergency.htm>
683. McGlown KJ, Fottler MD. The impact of flooding on the delivery of hospital services in the southeastern United States. *Health Care Manage Rev* 1996; 21: 55-71.
684. Fisher, HL. Emergency evacuation of the Denver Veteran's Administration Medical Center. *Milit Med* 1986; 151: 154-161.
685. Peters, MS. Hospitals respond to water loss during the midwest floods of 1993: Preparedness and improvisation. *J Emerg Med* 1996; 14: 345-350.
686. Joint Commission on Accreditation of Healthcare Organizations. *Comprehensive Accreditation Manual for Hospitals: The Official Handbook* (CAH00SJ). Oakbrook Terrace IL; JCAHO Press; 2000.
687. Tokars JI, Miller ER, Alter MJ, Arduino MJ. National Surveillance of Dialysis-Associated Diseases in the United States, 1997. Centers for Disease Control and Prevention, Public Health Service; U.S. Department of Health and Human Services; Atlanta, GA: 1998.
688. Stout JE, Best ME, Yu VL. Susceptibility of members of the family *Legionellaceae* to thermal stress: Implications for heat eradication methods in water distribution systems. *Appl Environ Microbiol* 1986; 52: 396-399.
689. Bornstein N, Vieilly C, Nowiki M, Paucod JC, Fleurette J. Epidemiological evidence of legionellosis transmission through domestic hot water supply systems and possibilities of control. *Isr J Med Sci* 1986; 13: 39-40.
690. Meenhorst PL, Reingold AL, Groothuis DG, et al. Water-related nosocomial pneumonia caused by *Legionella*

pneumophila serogroups 1 and 10. *J Infect Dis* 1985; 152: 356-364.

691. Mandel AS, Sprauer MA, Sniadack DH, Ostroff SM. State regulation in hospital water temperature. *Infect Control Hosp Epidemiol* 1993; 14: 642-645.

692. Department of Health. The control of *Legionella* in health care premises: A code of practice. London: HMSO, 1991.

693. Helms CM, Massanari RM, Wenzel RP, et al. Legionnaires' disease associated with a hospital water system: A five-year progress report on continuous hyperchlorination. *JAMA* 1988; 259: 2423-2427.

694. Edelstein PH, Whittaker RE, Kreiling RL, Howell, CL. Efficacy of ozone in eradication of *Legionella pneumophila* from hospital fixtures. *Appl Environ Microbiol* 1982; 44: 1330-1334

695. Muraca P, Stout JE, Yu, VL. Comparative assessment of chlorine, heat, ozone, and UV light for killing *Legionella pneumophila* within a model plumbing system. *Appl Environ Microbiol* 1987; 53: 447-453.

696. Domingue EL, Tyndall RL, Mayberry WR, Pancorbo OC. Effects of three oxidizing biocides of *Legionella pneumophila* serogroup 1. *Appl Environ Microbiol* 1988; 54: 741-747.

697. Landeen LK, Yahya MT, Gerba CP. Efficacy of copper and silver ions and reduced levels of free chlorine in inactivation of *Legionella pneumophila*. *Appl Environ Microbiol* 1989; 55: 3045-3050.

698. Matulonis U, Rosenfeld CS, Shaddock RK. Prevention of *Legionella* infections in bone marrow transplant unit: Multifaceted approach to decontamination of a water system. *Infect Control Hosp Epidemiol* 1993; 14: 571-583.

699. Liu Z, Stout JE, Tedesco L, et al. Controlled evaluation of copper-silver ionization in eradicating *Legionella pneumophila* from a hospital water distribution system. *J Infect Dis* 1994; 169: 919-922.

700. Margolin AB. Control of microorganisms in source water and drinking water. In: *Manual of Environmental Microbiology*, Hurst CJ, Knudsen GR, McInerney MJ, Stetzenback LD, Walter MV, eds. Washington DC; American Society for Microbiology Press; 1997: p. 195-202.

701. Freije MR. *Legionella Control in Health Care Facilities, A Guide for Minimizing Risk*. HC Information Resources, Inc. 1996: p. 65-75.

702. Yu-sen E, Lin R, Vidic D, Stout JE, Yu VL. *Legionella* in water distribution systems. *JAWWA* 1998; 90: 112-121.

703. Biurrun A, Caballero L, Pelaz C, Leon E, Gago A. Treatment of a *Legionella pneumophila*-colonized water distribution system using copper-silver ionization and continuous chlorination. *Infect Control Hosp Epidemiol* 1999; 20: 426-428.

704. Goetz A, Yu VL. Copper-silver ionization: Cautious optimism for *Legionella* disinfection and implications for environmental culturing. *Am J Infect Control* 1997; 25: 449-451.

705. Stout JE, Lin YS, Goetz AM, Muder RR. Controlling *Legionella* in hospital water systems: Experience with the superheat-and-flush method and copper-silver ionization. *Infect Control Hosp Epidemiol* 1998; 19: 911-914.

706. Rohr U, Senger M, Selenka F, Turley R, Wilhelm M. Four years of experience with silver-copper ionization for control of *Legionella* in a German university hospital hot water plumbing system. *Clin Infect Dis* 1999; 29: 1507-1511.

707. Cunliffe DA. Inactivation of *Legionella pneumophila* by monochloramine. *J Appl Bacteriol* 1990; 68: 453-459.

708. Kirmeyer GJ, Foust GW, Pierson GL, Simmler JJ, LeChevalier MW. *Optimizing Chloramine Treatment*. Denver CO; American Water Works Research Foundation; 1993.

709. Kool JL, Carpenter JC, Fields BS. Effect of monochloramine disinfection of municipal drinking water on risk of nosocomial Legionnaires' disease. *Lancet* 1999; 353: 272-277.

710. Kool JL, Bergmire-Sweet D, Butler JC, et al. Hospital characteristics associated with colonization of water systems by *Legionella* and risk of nosocomial Legionnaires' disease: A cohort study of 15 hospitals. *Infect Control Hosp Epidemiol* 1999; 20: 798-805.

711. Yu VL. Routine culturing for *Legionella* in the hospital environment may be a good idea: A three-hospital prospective study. *Am J Med* 1987; 294: 97-99

712. Allegheny County Health Department. Approaches to prevention and control of *Legionella* infection in Allegheny County health care facilities. Pittsburgh: Allegheny County Health Department, 1997: 1-13

713. Goetz AM, Stout JE, Jacobs SL, et al. Nosocomial Legionnaires' disease discovered in community hospitals following cultures of the water system: Seek and ye shall find. *Am J Infect Control* 1998; 26: 8-11.

714. Maryland Department of Health and Mental Hygiene. Report of the Maryland Scientific Working Group to Study *Legionella* in the Water Systems in Healthcare Institutions. June 14, 2000.

<http://www.dhmd.state.md.us/html/legionella.htm>

715. Yu VL. Nosocomial legionellosis: Current epidemiologic issues. In: *Current Clinical Topics in Infectious Diseases*. Remington JS, Swartz MN, eds. New York NY; McGraw-Hill; 1986: p. 239-253.

716. Vickers RM, Yu VL, Hanna SS. Determinants of *Legionella pneumophila* contamination of water distribution systems: 15-hospital prospective study. *Infect Control* 1987; 8: 357-363.
717. Tobin JO, Swann RA, Bartlett CLR. Isolation of *Legionella pneumophila* from water systems: Methods and preliminary results. *Br Med J* 1981; 282: 515-517.
718. Marrie TJ, Bezanson G, Fox J, Kuehn R, Haldane D, Birbridge S. Dynamics of *Legionella pneumophila* in the potable water of one floor of a hospital. In: *Legionella: Current Status and Emerging Perspectives*. Barbaree JM, Breiman RF, Dufow AP, eds. Washington DC; American Society for Microbiology Press; 1993: p, 238-240.
719. Plouffe JF, Para MF, Maher WE, Hackman B, Webster L. Subtypes of *Legionella pneumophila* serogroup 1 associated with different attack rates. *Lancet* 1983; 2: 649-650.
720. Fraser DW. Sources of legionellosis. In: *Legionella: Proceedings of the 2nd International Symposium*, Thornsberry C, Balows A, Feeley JC, Jakubowski W, eds. Washington DC; American Society for Microbiology Press; 1994: p. 277-280.
721. Dourmon E, Bibb WF, Rajagopalan P, Desplaces N, McKinney RM. Monoclonal antibody reactivity as a virulence marker for *Legionella pneumophila* serogroup 1 strain. *J Infect Dis* 1992; 165: 569-573.
722. Brundrett GW. Guides on avoiding Legionnaires' disease. In: *Legionella and Building Services*. Oxford UK; Butterworth Heineman; 1992: 346-373.
723. Kugler JW, Armitage JO, Helms CM, et al. Nosocomial Legionnaires' disease: Occurrence in recipients of bone marrow transplants. *Am J Med* 1983; 74: 281-288.
724. Lepine LA, Jernigan DB, Butler JC, et al. A recurrent outbreak of nosocomial Legionnaires' disease detected by urinary antigen testing: Evidence for long-term colonization of a hospital plumbing system. *Infect Control Hosp Epidemiol* 1998; 19: 905-910.
725. Barbaree JM. Selecting a subtyping technique for use in investigations of legionellosis epidemics. In: *Legionella: Current Status and Emerging Perspectives*, Barbaree JM, Breiman RF, Dufow AP, eds. Washington DC; American Society for Microbiology Press; 1993: .
726. Joly JR, McKinney RM, Tobin JO, Bibb WF, Watkins ID, Ramsay D. Development of a standardized subgrouping scheme for *Legionella pneumophila* serogroup 1 using monoclonal antibodies. *J Clin Microbiol* 1986; 23: 768-771.
727. Schoonmaker D, Helmberger T, Birkhead G. Comparison of ribotyping and restriction enzyme analysis using pulsed-field gel electrophoresis for distinguishing *Legionella pneumophila* isolates obtained during a nosocomial outbreak. *J Clin Microbiol* 1992; 30:1491-1498.
728. Johnston JM, Latham RH, Meier FA, et al. Nosocomial outbreak of Legionnaires' disease: Molecular epidemiology and disease control measures. *Infect Control* 1987; 8: 53-58.
729. Best MG, Goetz A, Yu VL. Heat eradication measures for control of nosocomial Legionnaires' disease: Implementation, education, and cost analysis. *Infect Control* 1984; 12: 26-30.
730. Muraca PW, Yu VL, Goetz A. Disinfection of water distribution systems for *Legionella*: A review of application procedures and methodologies. *Infect Control Hosp Epidemiol* 1990; 11: 79-88.
731. U.S. Department of Labor, Occupational Safety and Health Administration. OSHA Technical Manual, Section III, Chapter 7. Legionellosis. http://www.osha-slc.gov/dts/osta/otm/otm_iii/otm_iii_7.html
732. Rudnick JR, Beck-Sague CM, Anderson RL, Schable B, Miller JM, Jarvis WR. Gram-negative bacteremia in open-heart surgery patients traced to probable tap-water contamination of pressure-monitoring equipment. *Infect Control Hosp Epidemiol* 1996; 17: 281-285.
733. Miller RP. Cooling towers and evaporative condensers. *Ann Intern Med* 1979; 90: 667-670.
734. Butler JC, Breiman RF. Legionellosis. In: *Bacterial Infections of Humans*, 3rd ed., Evans AS, Brachman PS, eds. New York NY; Plenum Medical; 1998: p. 355-376.
735. Witherell LE, Novick LF, Stone KM, et al. *Legionella* in cooling towers. *J Environ Health* 1986; 49: 134-139.
736. Cordes LG, Fraser DW, Skaliy P, et al. Legionnaires' disease outbreak at an Atlanta, Georgia country club: Evidence for spread from an evaporative condenser. *Am J Epidemiol* 1980; 111: 425-431.
737. Kaufmann AF, McDade JE, Patton CM, et al. Pontiac fever: Isolation of the etiologic agent (*Legionella pneumophila*) and demonstration of its mode of transmission. *Am J Epidemiol* 1981; 114: 337-347.
738. Morton S, Bartlett CLR, Bibby LF, Hutchinson DM, Dyer JV, Dennis PJ. Outbreak of Legionnaires' disease from a cooling water system in a power station. *Br J Indust Med* 1986; 43: 630-635.
739. Friedman S, Spitalny K, Barbaree J, Faur Y, McKinney R. Pontiac fever outbreak associated with a cooling tower. *Am J Public Health* 1987; 77: 568-572.
740. Addiss DG, Davis JP, LaVenture M, Wand PJ, Hutchinson MA, McKinney RM. Community-acquired

Legionnaires' disease associated with a cooling tower: Evidence for longer-distance transport of *Legionella pneumophila*. *Am J Epidemiol* 1989; 130: 557-568.

741. Keller DW, Hajjeh R, DeMaria A Jr, et al. Community outbreak of Legionnaires' disease: An investigation confirming the potential for cooling towers to transmit *Legionella* species. *Clin Inf Dis* 1996; 22: 257-261.

742. Pastoris MC, Ciceroni L, Lo Monaco R, et al. Molecular epidemiology of an outbreak of Legionnaires' disease associated with a cooling tower in Genova-Sestri Ponente, Italy. *Eur J Clin Microbiol Infect Dis* 1997; 16: 883-892.

743. Brown CM, Nuorti PJ, Breiman RF, et al. A community outbreak of Legionnaires' disease linked to hospital cooling towers: An epidemiological method to calculate dose of exposure. *Inter J Epidemiol* 1999; 28: 353-359.

744. Broadbent CR. *Legionella* in cooling towers: Practical research, design, treatment, and control guidelines. In: *Legionella: Current Status and Emerging Perspectives*, Barbaree JM, Breiman RF, Dufour AP, eds. Washington DC; American Society for Microbiology Press; 1993: p. 217-222.

745. Bhopal RS, Barr G. Maintenance of cooling towers following two outbreaks of Legionnaires' disease in a city. *Epidemiol Infect* 1990; 104: 29-38.

746. Centers for Disease Control. Suggested health and safety guidelines for public spas and hot tubs. Publication No. 99-960, Centers for Disease Control, Atlanta, GA; 1985.

747. World Health Organization. *Environmental Aspects of the Control of Legionellosis*, 14th ed. Copenhagen Denmark; World Health Organization; 1986: p. 118-120.

748. World Health Organization. Epidemiology, prevention, and control of legionellosis: Memorandum from a WHO meeting. *Bull WHO* 1990; 68: 155-164.

749. Association for the Advancement of Medical Instrumentation. American National Standard *Hemodialysis Systems* ANSI/AAMI RD5-1981, Association for the Advancement of Medical Instrumentation, Arlington, VA 1982.

750. Association for the Advancement of Medical Instrumentation. American National Standard *Hemodialysis Systems* ANSI/AAMI RD5-1992, Association for the Advancement of Medical Instrumentation, Arlington, VA 1993.

751. Association for the Advancement of Medical Instrumentation. *Reuse of Hemodialyzers* ROH-1986, Association for the Advancement of Medical Instrumentation, Arlington, VA 1986.

752. Association for the Advancement of Medical Instrumentation. American National Standard *Reuse of Hemodialyzers* ANSI/AAMI RD47-1993, Association for the Advancement of Medical Instrumentation, Arlington, VA 1993.

753. Tokars JI, Miller ER, Alter MJ, Arduino MJ. National surveillance of dialysis associated diseases in the United States, 1997. *Seminars in Dialysis* 2000; 13: 75-85.

754. Hindman SH, Carson LA, Petersen NJ, et. al. Pyrogenic reactions during hemodialysis caused by extramural endotoxin. *Lancet* 1975; 2: 732-734.

755. Stamm JE, Engelhard WE, Parson JE. Microbiological study of water softener resins. *Appl Microbiol* 1969; 18: 376-386.

756. Alter MJ, Favero MS, Miller JK, Coleman BJ, Bland LA. National surveillance of dialysis-associated diseases in the United States, 1988. *ASAIO Trans* 1990; 36: 107-118.

757. Tokars JI, Alter MJ, Favero MS, Moyer LA, Bland LA. National surveillance of dialysis- associated diseases in the United States, 1990. *ASAIO J* 1993; 39: 71-80.

758. Tokars JI, Alter MJ, Favero MS, Moyer LA, Bland LA. National surveillance of dialysis- associated diseases in the United States, 1991. *ASAIO J* 1993; 39: 966-975.

759. Tokars JI, Alter MJ, Favero MS, Moyer LA, Bland LA. National surveillance of dialysis- associated diseases in the United States, 1993. *ASAIO J* 1996; 42: 219-229.

760. Petersen NJ, Boyer KM, Carson LA, Favero MS. Pyrogenic reactions from inadequate disinfection of a dialysis unit distribution system. *Dialysis Transpl* 1978; 7: 52-57.

761. Gazenfeldt-Gazit E, Elaihou HE. Endotoxin antibodies in patients on maintenance hemodialysis. *Israel J Med Sci* 1969; 5:1032-1036.

762. Laude-Sharp M, Canoff M, Simard L, Pusineri C, Kazatchkine M, Haeffner-Cavaillon N. Induction of IL-1 during hemodialysis: Transmembrane passage of intact endotoxin (LPS). *Kidney Int* 1990; 38:1089-1094.

763. Arduino MJ, Bland LA, McAllister SK, Favero MS. The effects of endotoxin contaminated dialysate and polysulfone or cellulosic membranes on the release of TNF α during simulated dialysis. *Artif Organs* 1995; 19: 880-886.

764. Greisman SE, Hornick RB. Comparative pyrogenic reactivity of rabbit and man to bacterial endotoxin. *Proc Soc Exp Biol Med* 1969; 131: 1154-1158.

765. Weary ME, Donohue G, Pearson FC, Story K. Relative potencies of four reference endotoxin standards as measured by the Limulus amoebocyte lysate and USP rabbit pyrogen tests. *Appl Environ Microbiol* 1980; 40: 1148-1151.

766. Bland LA, Ridgeway MR, Aguero SM, Carson LA, Favero MS. Potential bacteriologic and endotoxin hazards associated with liquid bicarbonate concentrate. *ASAIO Trans* 1987; 33: 542-545.
767. Dawids SG, Vejlsgaard R. Bacteriological and clinical evaluation of different dialysate delivery systems. *Acta Med Scand* 1976; 199: 151-155.
768. Favero MS, Alter MJ, Tokars JI, Arduino MJ. Dialysis-associated infections and their control; In: *Hospital Infections* 4th Ed., Bennett JV, Brachman PS, eds. Philadelphia PA; Lippincott-Raven; 1998: p. 357-380.
769. Kidd EE. Bacterial contamination of dialyzing fluid of artificial kidney. *Brit Med J* 1964; 880-882.
770. Jones DM, Tobin BM, Harlow GR, et al. Bacteriological studies of the modified kiil dialyzer. *Brit Med J* 1970; 3: 135-137.
771. Raij L, Shapiro FL, Michael AF. Endotoxemia in febrile reactions during hemodialysis. *Kidney Int* 1973; 4: 57-60.
772. Vanholder R, Van Haecke E, Veys N, et al. Endotoxin transfer through dialysis membranes: Small versus large-pore membranes. *Nephrol Dial Transplant* 1992; 7: 333-339.
773. Evans RC, Holmes CJ. *In vitro* study of the transfer of cytokine-inducing substances across selected high-flux hemodialysis membranes. *Blood Purif* 1991; 9: 92-101.
774. Lonnemann G, Behme TC, Lenzer B, et al. Permeability of dialyzer membranes to TNF^α-inducing substances derived from water bacteria. *Kidney Int* 1992; 42: 61-68.
775. Ureña P, Herbelin A, Zingraff J, et al. Permeability of cellulosic and non-cellulosic membranes to endotoxin subunits and cytokine production during *in-vitro* hemodialysis. *Nephrol Dial Transplant*. 1992; 7:1628.
776. Bommer J, Becker KP, Urbaschek R. Potential transfer of endotoxin across high-flux polysulfone membranes. *J Amer Soc Nephrol* 1996; 7: 883-888.
777. Yamagami S, Adachi T, Sugimura, T, et al. Detection of endotoxin antibody in long-term dialysis patients. *Int J Artif Organs* 1990; 13: 205-210.
778. Arduino MJ. CDC investigations of noninfectious outbreaks of adverse events in hemodialysis facilities, 1979-1999. *Seminars Dialysis* 2000; 13: 86-91.
779. Roth V, Jarvis WR. Outbreaks of infection and/or pyrogenic reactions in dialysis patients. *Seminars Dialysis* 2000; 13: 92-100.
780. Gordon SM, Tipple MME, Bland LA, Jarvis WR. Pyrogenic reactions associated with reuse of disposable hollow-fiber hemodialyzers. *JAMA* 1988; 260: 2077-2081.
781. Alter MJ, Tokars JI, Arduino MJ. Nosocomial infections in hemodialysis units - Strategies for control. In: *Dialysis and Transplantation: A Companion to Brenner and Rector's "The Kidney,"* Owen WF, Periera BJG, Sayegh MH, eds. Orlando FL; WB Saunders Company; 1999.
782. Bernick JJ, Port FK, Favero MS, Brown DG. Bacterial and endotoxin permeability of hemodialysis membranes. *Kidney Int* 1979; 16: 491-496.
783. Bommer J, Becker KP, Urbaschek R, Ritz E, Urbaschek B. No evidence for endotoxin transfer across high flux polysulfone membranes. *Clin Nephrol* 1987; 27: 278-282.
784. Schindler R, Lonnemann G, Schaeffer J, et al. The effect of ultrafiltered dialysate on the cellular content of interleukin-1 receptor antagonist in patients on chronic hemodialysis. *Nephron* 1994; 68: 229-233.
785. Akrum RAE, Frolich M, Gerritsen AF, et al. Improvement of chronic inflammatory state in hemodialysis patients by the use of ultrapure water for dialysate. *J Amer Soc Nephrol* 1997; 8: 226A.
786. Quellhorst E. Methods of Hemodialysis. *Nieren U Hochdruck* 1998; 27: 35-41.
787. Baz M, Durand C, Ragon A, et al. Using ultrapure water in hemodialysis delays carpal tunnel syndrome. *Int J Artif Organs* 1991; 14: 681-685.
788. Schwalbe S, Holzhauer M, Schaeffer J, et al. β_2 -Microglobulin associated amyloidosis: A vanishing complication of long-term hemodialysis? *Kidney Int* 1997; 52: 1077-1083.
789. Arduino MJ, Favero MS. Microbiologic aspects of hemodialysis. *Water Quality for Hemodialysis*. AAMI Monograph WQD-1998; Arlington VA; Association for the Advancement of Medical Instrumentation; 1998.
790. Leyboldt JK, Schmidt B, Gurland, HJ. Measurement of backfiltration rates during hemodialysis with highly permeable membranes. *Blood Purif* 1991; 9: 74-84.
791. Carson LA, Bland LA, Cusick LB, Collin S, Favero MS, Bolan G. Factors affecting endotoxin levels in fluids associated with hemodialysis procedures. In: *Detection of Bacterial Endotoxins with the Limulus Amoebocyte Lysate Test*, Novitsky TJ, Watson SW, eds. New York NY; Alan R. Liss; 1987: p. 223-234.
792. Anderson RL, Holland BW, Carr JK, Bond WW, Favero MS. Effect of disinfectants on pseudomonads colonized on the interior surface of PVC pipes. *Am J Public Health* 1990; 80: 17-21.

793. Bland LA, Favero MS. Microbial contamination control strategies for hemodialysis. *JCAHO Plant Tech Manage Series* 1989; 3: 30-36.
794. Bland LA. Microbiological and endotoxin assays of hemodialysis fluids. *Adv Renal Replacement Ther* 1995; 2: 70-79.
795. Arduino MJ, Bland LA, Agüero SM, Carson LA, Ridgeway M, Favero MS. Comparison of microbiologic assay methods for hemodialysis fluids. *J Clin Microbiol* 1991; 29: 592-594.
796. Association for the Advancement of Medical Instrumentation. American National Standard Water Treatment Equipment for Hemodialysis Applications. ANSI/AAMI RD62-199X. Arlington VA; Association for the Advancement of Medical Instrumentation: (1999 Draft).
797. Arduino MJ. How should dialyzers be reprocessed? *Seminars in Dialysis* 1998; 11: 282-284.
798. Jochimsen EM, Frenette C, Delorme M, et al. A cluster of bloodstream infections and pyrogenic reactions among hemodialysis patients traced to dialysis machine waste-handling option units. *Am J Nephrol* 1998; 18: 485-489.
799. Wang SA, Levine RB, Carson LA, et al. An outbreak of gram-negative bacteremia in hemodialysis patients traced to hemodialysis machine waste drain ports. *Infect Control Hosp Epidemiol* 1999; 20: 746-751.
800. National Institutes of Health. *U.S. Renal Diseases Survey: 1999 Annual Data Report*. Bethesda MD; National Institute of Diabetes, Digestive and Kidney Diseases; Division of Kidney, Urologic, and Hematologic Diseases; 1999.
801. Monsen T, Olofson C, Ronnmark M, Wistrom J. Clonal spread of staphylococci among patients with peritonitis associated with continuous ambulatory peritoneal dialysis. *ASAIO J* 2000; 57: 613-618.
802. Band JD, Ward JJ, Fraser DW, et al. Peritonitis due to a *Mycobacterium chelonae* - like organism associates with intermittent chronic peritoneal dialysis. *J Infect Dis* 1982; 145: 9-17.
803. Monsen T, Crabtree JH, Siddiqui RA, et al. Dialysis catheter infection related peritonitis: Incidence and time dependent risk. *ASAIO J* 1999; 45: 574-580.
804. Vera G, Lew SQ. *Mycobacterium fortuitum* peritonitis in two patients receiving continuous ambulatory peritoneal dialysis. *Am J Nephrol* 1999; 19: 586-589.
805. Soriano F, Rodriguez-Tudela JL, Gomez-Garces JL, Velo M. Two possibly related cases of *Mycobacterium fortuitum* peritonitis in continuous ambulatory peritoneal dialysis. *Eur J Clin Microbiol* 1989; 8: 895-897.
806. Szeto CC, Li PK, Leung CB, Yu AW, Lui SF, Lai NK. *Xanthomonas maltophilia* peritonitis in uremic patients receiving ambulatory peritoneal dialysis. *Am J Kidney Dis* 1997; 29: 991-995.
807. Panlilio AL, Beck-Sague CM, Siegel JD, et al. Infections and pseudoinfections due to povidone-iodine solution contaminated with *Pseudomonas cepacia*. *Clin Infect Dis* 1992; 14: 1078-1083.
808. Riebel W, Frantz N, Adelstein D, Spanguolo PJ. *Corynebacterium JK*: A cause of nosocomial device-related infection. *Rev Infect Dis* 1986; 8: 42-49.
809. Radix AE, Bieluch VM, Graeber CW. Peritonitis caused by *Monilia sitophila* in a patient undergoing peritoneal dialysis. *Int J Artif Organs* 1996; 19: 218-220.
810. Banerjee S, Marwaha RK, Bajwa RP. Fungal peritonitis complicating peritoneal dialysis. *Indian Pediatr* 1995; 32: 693-697.
811. Bergeson E, Denis R, Cartier P. Peritoneal dialysis: Peritonitis and catheter infections. *Annales de Chirurgie* 1996; 50: 606-612. (French)
812. Troidle L, Kligler AS, Goldie SJ, et al. Continuous peritoneal dialysis-associated peritonitis of nosocomial origin. *Perit Dialysis International* 1996; 16: 505-510.
813. Smith CA. Reduced incidence of peritonitis by utilizing "flush before fill" in APD. *Adv Perit Dialysis* 1997; 13: 224-226.
814. Valeri A, Radhakrishnan J, Vernocchi L, Carmichael LD, Stern L. The epidemiology of peritonitis in acute peritoneal dialysis: A comparison between open- and closed drainage systems. *Am J Kidney Dis* 1993; 21: 300-309.
815. Stamm WE, Colelle JJ, Anderson RL, Dixon RE. Indwelling arterial catheters as a source of nosocomial bacteremia. An outbreak caused by *Flavobacterium* species. *N Engl J Med* 1975; 292: 1099-1102.
816. Schimpff SC. Gram negative bacteremia. *Support Care Cancer* 1993; 1: 5-18.
817. Graman PS, Quinlan GA, Rank JA. Nosocomial legionellosis traced to contaminated ice. *Infect Control Hosp Epidemiol* 1997; 18: 637-640.
818. Gahrn-Hansen B, Uldum SA, Schmidt J, Nielsen B, Birkeland SA, Jorgensen KA. [Nosocomial *Legionella pneumophila* infection in a nephrology department]. *Ugeskrift for Laeger* 1995; 157: 590-594. (German)
819. Wilson IG, Hogg GM, Barr JG. Microbiological quality of ice in hospital and community. *J Hosp Infect* 1997; 36: 171-180.
820. Spencer RC. The emergence of epidemic, multiple-antibiotic-resistant *Stenotrophomonas (Xanthomonas)*

maltophilia and *Burkholderia (Pseudomonas) cepacia*. *J Hosp Infect* 1995; 30 (Suppl): 453-464.

821. Cannon RO, Poliner JR, Hirschhorn RB, et al. A multistate outbreak of Norwalk virus gastroenteritis associated with consumption of commercial ice. *J Infect Dis* 1991; 164: 860-863.

822. Khan AS, Moe CL, Glass RI, et al. Norwalk virus-associated gastroenteritis traced to ice consumption aboard a cruise ship in Hawaii: Comparison and application of molecular method-based assays. *J Clin Microbiol* 1994; 32: 318-322.

823. Centers for Disease Control and Prevention. Outbreak of viral gastroenteritis - Pennsylvania and Delaware. *MMWR* 1987; 36: 709-711.

824. Quick R, Paugh K, Addiss D, Kobayashi J, Baron R. Restaurant-associated outbreak of giardiasis. *J Infect Dis* 1992; 166: 673-676.

825. Hedberg CW, White KE, Johnson JA, et al. An outbreak of *Salmonella enteritidis* infection at a fast food restaurant: Implications for foodhandler-associated transmission. *J Infect Dis* 1991; 164: 1135-1140.

826. Burnett IA, Weeks GR, Harris DM. A hospital study of ice-making machines: Their bacteriology, design, usage, and upkeep. *J Hosp Infect* 1994; 28: 305-313.

827. Petersen NJ. "Don't culture the ice machines." *Hosp Infect Control* 1982; 9: 8-9.

828. Centers for Disease Control. Sanitary care and maintenance of ice chests and ice machines. Atlanta, GA: CDC; 1979. No. 00-2384.

829. Manangan LP, Anderson RL, Arduino MJ, Bond WW. Sanitary care and maintenance of ice-storage chests and ice-making machines in healthcare facilities. *Am J Infect Control* 1998; 26: 111-112.

830. Anonymous. Ice as a source of infection. *CDR Weekly* 1993; 3: 241.

831. Cardaney CR, Rodeheaver GT, Horowitz, JH, Kenney JG, Edlich RF. Influence of hydrotherapy and antiseptic agents on burn wound bacteria contamination. *J Burn Care Rehab* 1985; 6: 230-232.

832. Gruber RP, Laub DR, Vistnes LM. The effect of hydrotherapy on the clinical course and pH of experimental cutaneous chemical burns. *Plastic Reconstruct Surg* 1975; 55: 200-204.

833. Mansell RE, Borchardt KA. Disinfecting hydrotherapy equipment. *Arch Phys Med Rehabil* 1974; 55: 318-320.

834. Hall J, Skevington SM, Maddison PH, Chapman K. A randomized and controlled trial of hydrotherapy in rheumatoid arthritis. *Arthritis Care Res* 1996; 9: 206-215.

835. Gross A, Cutright DE, Bhaskar SN. Effectiveness of pulsating water jet lavage in treatment of contaminated crush injuries. *Am J Surgery* 1972; 124: 373-377.

836. Rodeheaver GT, Paltry D, Thacker JG, Edgerton MT, Edlich RF. Wound cleansing by high pressure irrigation. *Surg Gynecol Obstetr* 1975; 141: 357-362.

837. Saxe A, Goldstein E, Dixon S, Ostrup R. Pulsatile lavage in the management of postoperative wound infections. *Am Surgeon* 1980; 46: 391-397.

838. Weller K. In search of efficacy and efficiency: An alternative to conventional wound cleansing modalities. *Ostomy/Wound Manage* 1991; 37: 23-28.

839. Hicks CB, Chulay JD. Bacteremic *Citrobacter freundii* cellulitis associated with tub immersion in a patient with the nephrotic syndrome. *Mil Med* 1988; 153: 400-401.

840. Mayhall CG, Lamb VA, Gayle WE, Haynes BW. *Enterobacter cloacae* septicemia in a burn center: Epidemiology and control of an outbreak. *J Infect Dis* 1979; 139: 166-171.

841. Marrie TJ, Gass RSR, Yates L. *Legionella pneumophila* in a physiotherapy pool. *Eur J Clin Microbiol* 1987; 6: 212-213.

842. Havelaar AH, Berwald LG, Groothuis DG, Baas JG. Mycobacteria in semi-public swimming pools and whirlpools. *Ztb Bakteriolog Mikrobiol Hyg [B]* 1985; 180: 505-514.

843. Favero MS. Whirlpool spa-associated infections: Are we really in hot water? *Am J Public Health* 1984; 74: 653-655.

844. Ratnam S, Hogan K, March SB, Butler RW. Whirlpool-associated folliculitis caused by *Pseudomonas aeruginosa*: Report of an outbreak and review. *J Clin Microbiol* 1986; 23: 655-659.

845. Stone HH, Kolb LD. The evolution and spread of gentamicin-resistant pseudomonads. *J Trauma* 1971; 11: 586-589.

846. Richard P, LeFlock R, Chamoux C, Pannier M, Espaze E, Richet H. *Pseudomonas aeruginosa* outbreak in a burn unit: Role of antimicrobials in the emergence of multiply resistant strains. *J Infect Dis* 1994; 170: 377-383.

847. Schmidt OW, Cooney MK, Foy HM. Adeno-associated virus in adenovirus type 3 conjunctivitis. *Infection Immunity* 1975; 11: 1362-1370.

848. Solomon SL. Host factors in whirlpool-associated *Pseudomonas aeruginosa* skin disease. *Infect Control* 1985;

6: 402-406.

849. DeJonckheere JF. Hospital hydrotherapy pools treated with ultraviolet light: Bad bacteriological quality and presence of thermophilic *Naegleria*. *J Hyg (Lond)* 1982; 88: 205-214.
850. American Physical Therapy Association. Hydrotherapy/Therapeutic Pool Infection Control Guidelines. Alexandria VA; APTA; 1995: P-112
851. Centers for Disease Control. Disinfection of Hydrotherapy Pools and Tanks. 1974; HHS 00-2383. Atlanta, GA. Centers for Disease Control, Public Health Service, U.S. Department of Health and Human Services.
852. Price D, Ahearn DG. Incidence and persistence of *Pseudomonas aeruginosa* in whirlpools. *J Clin Microbiol* 1988; 26: 1650-1654.
853. Mayhall CG. Infections in burn patients. In: *APIC Infection Control and Applied Epidemiology: Principles and Practices*, Olmsted R, ed. St. Louis, MO; Mosby Year Book Publications; 1996: p. 44: 1-7.
854. Smith RF, Blasi D, Dayton SL, Chipps DD. Effects of sodium hypochlorite on the microbial flora of burns and normal skin. *J Trauma* 1974; 14: 938-944.
855. Cardany CR, Rodeheaver GT, Horowitz JH, Kenney JG, Edlich RF. Influence of hydrotherapy and antiseptic agents on burn wound bacterial contamination. *J Burn Care Rehabil* 1985; 6: 230-232.
856. Steve L, Goodhart P, Alexander J. Hydrotherapy burn treatment: Use of chloramine-T against resistant microorganisms. *Arch Phys Med Rehabil* 1979; 60: 301-303.
857. Golland A. Basic hydrotherapy. *Physiotherapy* 1981; 67: 258-262.
858. Edlich RF, Becker DG, Phung D, McClelland WA, Day SG. Water treatment of hydrotherapy exercise pools. *J Burn Care Rehabil* 1988; 9: 9510-9515.
859. Penny PT. Hydrotherapy pools of the future - The avoidance of health problems. *J Hosp Infect* 1991; 18: 535-542.
860. Centers for Disease Control. Swimming Pools: Safety and Disease Control Through Proper Design and Operation. 1976. HHS No. 88-8319. Atlanta GA; U.S. Department of Health and Human Services.
861. Linneman CC Jr. Nosocomial infections associated with physical therapy, including hydrotherapy, In: *Hospital Epidemiology and Infection Control*, 2nd Ed., Mayhall CG, ed. Philadelphia PA; Lippincott Williams & Wilkins; 1999: p. 931-936.
862. Aspinall ST, Graham R. Two sources of contamination of a hydrotherapy pool by environmental organisms. *J Hosp Infect* 1989; 14: 285-292.
863. McCandlish R, Renfrew M. Immersion in water during labor and birth: The need for evaluation. *Birth* 1993; 20: 79-85.
864. Hawkins S. Water vs conventional births: Infection rates compared. *Nursing Times* 1995; 91: 38-40.
865. Eriksson M, Ladfors L, Mattsson LA, Fall O. Warm tub bath during labor: A study of 1385 women with prelabor rupture of the membranes after 34 weeks of gestation. *Acta Obstet Gynaecol Scand* 1996; 75: 642-644.
866. Rush J, Burlock S, Lambert K, Loosley-Millman M, Hutchinson B, Enkin M. The effects of whirlpool baths in labor: A randomized, controlled trial. *Birth* 1996; 23: 136-143.
867. Davis BJ. Whirlpool operation and the prevention of infection. *Infect Control* 1985; 6: 394-397.
868. Babb JR, Bradley CR, Ayliffe GAJ. Comparison of automated systems for the cleaning and disinfection of flexible fiberoptic endoscopes. *J Hosp Infect* 1984; 5: 213-226.
869. Babb JR, Bradley CR. The mechanics of endoscope disinfection. *J Hosp Infect* 1991; 18 (Suppl A): 130-135.
870. Gubler JGH, Salfinger M, von Graevenitz A. Pseudoepidemic of nontuberculous mycobacteria due to a contaminated bronchoscope cleaning machine. *Chest* 1992; 101: 1245-1249.
871. Fraser VJ, Jones M, Murray PR, Medoff G, Zhang Y, Wallace RJ Jr. Contamination of flexible fiberoptic bronchoscopes with *Mycobacterium chelonae* linked to an automated bronchoscope disinfection machine. *Am Rev Respir Dis* 1992; 145: 853-855.
872. Maloney S, Welbel S, Daves B, et al. *Mycobacterium abscessus* pseudoinfection traced to an automated endoscope washer: Utility of epidemiologic and laboratory investigation. *J Infect Dis* 1994; 169: 1166-1169.
873. Merighi A, Contato E, Scagliarini R, et al. Quality improvement in gastrointestinal endoscopy: Microbiologic surveillance of disinfection. *Gastrointest Endosc* 1996; 43: 457-462.
874. Griffiths PA, Babb JR, Bradley CR, Fraise AP. Glutaraldehyde-resistant *Mycobacterium chelonae* from endoscope washer disinfectors. *J Appl Microbiol* 1997; 82: 519-526.
875. Phillips G, McEwan H, Butler J. Quality of water in washer-disinfectors. *J Hosp Infect* 1995; 31: 152-154.
876. Cooke RPD, Whymant-Morris A, Umasankar RS, Goddard SV. Bacteria-free water for automatic washer-disinfectors: An impossible dream? *J Hosp Infect* 1998; 48: 63-65.

877. Humphreys H, Lee JV. Water quality for endoscopy washer-disinfectors. *J Hosp Infect* 1999; 42: 76-78.
878. Mitchell DH, Hicks LJ, Chiew R, Montanaro JC, Chen SC. Pseudoepidemic of *Legionella pneumophila* serogroup 6 associated with contaminated bronchoscopes. *J Hosp Infect* 1997; 37: 19-23.
879. Ido K, Ishino Y, Ota Y, et al. Deficiencies of automatic endoscopic reprocessors: a method to achieve high-grade disinfection of endoscopes. *Gastrointest Endosc* 1996; 44: 583-586.
880. Allen JJ, Allen MO, Olsen MM, et al. *Pseudomonas* infection of the biliary system resulting from the use of a contaminated endoscope. *Gastroenterology* 1987; 92: 759-763.
881. Agerton T, Valway S, Gore B, et al. Transmission of a highly drug-resistant strain (Strain W-1) of *Mycobacterium tuberculosis*: Community outbreak and nosocomial transmission via a contaminated bronchoscope. *JAMA* 1997; 278: 1073-1077
882. Michele TM, Cronin WA, Graham NMH, et al. Transmission of *Mycobacterium tuberculosis* by a fiberoptic bronchoscope: Identification by DNA fingerprinting. *JAMA* 1997; 278: 1093-1095.
883. Bronowicki J-P, Venard V, Botte C, et al. Patient-to-patient transmission of hepatitis C virus during colonoscopy. *N Engl J Med* 1997; 337: 237-240.
884. Alvarado CJ, Stolz SM, Maki DG. Nosocomial infections from contaminated endoscopes: A flawed automated endoscope washer. An investigation using molecular epidemiology. *Am J Med* 1991; 91 (Suppl 3b): 272-280.
885. Rey JF. Endoscopic disinfection. A worldwide problem. *J Clin Gastroenterol* 1999; 28: 291-297.
886. Alvarado CJ, Reichelderfer M. APIC guideline for infection prevention and control in flexible endoscopy. *Am J Infect Control* 2000; 28: 138-155.
887. Van Klingeren B, Pullen W. Glutaraldehyde resistant mycobacteria from endoscope washers. *J Hosp Infect* 1993; 25: 147-149.
888. Flournoy DJ, Petrone RL, Voth DW. A pseudo-outbreak of *Methylobacterium mesophilica* isolated from patients undergoing bronchoscopy. *Eur J Clin Microbiol Infect Dis* 1992; 11: 240-243.
889. Kelstrup J, Funder-Nielsen T, Theilade J. Microbial aggregate contamination of water lines in dental equipment and its control. *Acta Path Scand* 1977; 85: 177-183.
890. Challacombe SJ, Fernandes LL. Detecting *Legionella pneumophila* in water systems: A comparison of various dental units. *J Am Dent Assoc* 1995; 126: 603-608.
891. Centers for Disease Control and Prevention. Statement from the Centers for Disease Control and Prevention (CDC) Regarding Biofilm and Dental Unit Water Quality. 1999. Atlanta GA; U.S. Public Health Service; Department of Health and Human Services.
892. Centers for Disease Control and Prevention. Recommended infection control practices for dentistry, 1993. *MMWR* 1993; 42 (RR-8): 1-12.
893. Office of Safety and Asepsis Procedures Research Foundation. Position paper on dental unit waterlines. 2000; OSAP, P.O. Box 6297, Annapolis MD 21401.
894. U.S. Environmental Protection Agency. National Primary Drinking Water Regulations, 1999. At: <http://www.epa.gov/OGWDW/wot/appa.html>
895. American Public Health Association, American Water Works Association, Water Environment Foundation. *Standard Methods for the Examination of Water and Wastewater*, 20th Ed., Eaton AD, Clesceri LS, Greenberg AE, eds. Washington DC; American Public Health Association; 1999: p. 9-1 - 9-41.
896. Maki DG, Alvarado CJ, Hassemer CA, Zilz MA. Relation of the inanimate hospital environment to endemic nosocomial infection. *N Engl J Med* 1982; 307: 1562-1566.
897. Danforth D, Nicolle LE, Hume K, Alfieri N, Sims H. Nosocomial infections on nursing units with floors cleaned with a disinfectant compared with detergent. *J Hosp Infect* 1987; 10: 229-235.
898. Spaulding EH. Role of chemical disinfection in the prevention of nosocomial infections. In: *Proceedings of the International Conference on Nosocomial Infections, 1970*. Brachman PS, Eickhoff TC, eds. Chicago IL. American Hospital Association; 1971: p. 247-254.
899. Spaulding EH. Chemical disinfection and antisepsis in the hospital. *J Hosp Res* 1972; 9: 5-31.
900. Favero MS, Bond WW. Chemical disinfection of medical and surgical materials. In: *Disinfection, Sterilization, and Preservation*, 4th Ed. Block SS, ed. Philadelphia PA; Lea & Febiger; 1991: p.617-641.
901. Rutala WA. APIC guideline for selection and use of disinfectants. *Am J Infect Control* 1996; 24: 313-342.
902. Favero MS, Bond WW. Sterilization, disinfection, and antisepsis in the hospital. In: *Manual of Clinical Microbiology*, 5th Ed. Balows A, Hausler WJ Jr, Herrmann KL, Isenberg HD, Shadomy HJ, eds. Washington DC; American Society for Microbiology; 1991: p.183-200.
903. Nyström B. Bioburden of non-disposable surgical instruments and operating room textiles. In: *Sterilization of*

Medical Products, Vol II. Gaughran ERL, Morrissey RF, eds. Montreal Québec, Canada; Multiscience Publications Ltd; 1981; p. 156-163.

904. Nyström B. Disinfection of surgical instruments. *J Hosp Infect* 1981; 2: 3636-368.

905. Rutala WA, Weber DJ. FDA labeling requirements for disinfection of endoscopes: A counterpoint. *Infect Control Hosp Epidemiol* 1995; 16: 231-235.

906. Parker HH IV, Johnson RD. Effectiveness of ethylene oxide for sterilization of dental handpieces. *J Dent* 1995; 1: 1-3.

907. Alfa MJ, DeGagne P, Olson N, Puchalski T. Comparison of ion plasma, vaporized hydrogen peroxide, and 100% ethylene oxide sterilizers to the 12/88 ethylene oxide gas sterilizer. *Infect Control Hosp Epidemiol* 1996; 17: 92-100.

908. Rutala WA, Gergen MF, Jones JF, Weber DJ. Levels of microbial contamination on surgical instruments. *Am J Infect Control* 1998; 26: 143-145.

909. U.S. Food and Drug Administration (FDA) and U.S. Environmental Protection Agency (EPA). Memorandum of understanding between the Food and Drug Administration, Public Health Service, Department of Health and Human Services, and the Environmental Protection Agency: Notice regarding matters of mutual responsibility - regulation of liquid chemical germicides intended for use on medical devices, 1993. Available from FDA, Center for Devices and Radiological Health (CDRH), Office of Health and Industry Programs, Division of Small Manufacturers Assistance, Rockville MD 20850, or EPA, Registration Division, Antimicrobial Program Branch, 401 M St., SW, Washington DC 20460.

910. U.S. Food and Drug Administration (FDA). Interim measures for the registration of antimicrobial products/liquid chemical germicides with medical device use claims under the memorandum of understanding between EPA and FDA, 1994. CDRH Facts on Demand, Shelf #851, p. 14; June 30, 1994. Available from FDA, CDRH, Office of Health and Industry Programs, Division of Small Manufacturers Assistance, Rockville MD 20850.

911. U.S. Department of Labor, Occupational Safety and Health Administration. Occupational Exposure to Bloodborne Pathogens: Final rule (29 CFR 1910.1030). *Federal Register* 1991; 56: 64004-64182.

912. Collins BJ. The hospital environment: How clean should a hospital be? *J Hosp Infect* 1988; 11 (Suppl A): 53-56.

913. Van den Berg RWA, Claahsen HL, Niessen M, Muytjens HL, Liem K, Voss A. *Enterobacter cloacae* outbreak in the NICU related to disinfected thermometers. *J Hosp Infect* 2000; 45: 29-34.

914. Spaulding EH. Alcohol as a surgical disinfectant. *AORN J* 1964; 2: 67-71.

915. Ayliffe GAJ, Collins BJ, Lowbury EJJ, Babb JR, Lilly HA. Ward floors and other surfaces as reservoirs of hospital infection. *J Hyg (Camb)* 1967; 65: 515-537.

916. Dancer SJ. Mopping up hospital infection. *J Hosp Infect* 1999; 43: 85-100.

917. Gable TS. Bactericidal effectiveness of floor cleaning methods in a hospital environment. *Hospitals JAHA* 1966; 40: 107-111.

918. Petersen NJ, Marshall JH, Collins DE. Why wash walls in hospital isolation rooms? *Health Lab Sci* 1973; 10: 23-27.

919. Ayliffe GAJ, Collins BJ, Lowbury EJJ. Cleaning and disinfection of hospital floors. *Brit Med J* 1966; 2: 442-445.

920. Vesley D, Pryor AK, Walter WG, Shaffer JG. A cooperative microbiological evaluation of floor-cleaning procedures in hospital patient rooms. *Health Lab Sci* 1970; 7: 256-264.

921. Daschner J, Rabbenstein G, Langmaack H. [Fla chendekontamination zur verhütung und bekämpfung von drakenhaus infectionen.] *Deutsche Medizinische Wochenschrift* 1980; 10: 325-329. (German)

922. Palmer PH, Yeoman DM. A study to assess the value of disinfectants when washing ward floors. *Med J Australia* 1972; 2: 1237-1239.

923. Schmidt EA, Coleman DL, Mallison GF. Improved system for floor cleaning in health care facilities. *Appl Environ Microbiol* 1984; 47: 942-946.

924. Mallison GF. Hospital disinfectants for housekeeping: Floors and tables. *Infect Control* 1984; 5: 537.

925. Vesley D, Klapes NA, Benzow K, Le CT. Microbiological evaluation of wet and dry floor sanitization systems in hospital patient rooms. *Appl Environ Microbiol* 1987; 53: 1042-1045.

926. Werry C, Lawrence JM, Sanderson PJ. Contamination of detergent cleaning solutions during hospital cleaning. *J Hosp Infect* 1988; 11: 44-49.

927. Chou T. Environmental Services. In: *APIC Infection Control and Applied Epidemiology: Principles and Practice*. Olmstead RN, ed. St. Louis MO; Mosby-YearBook; 1996: p. 107.1-107.7.

928. Rutala WA, Shafer KM. General Information on Cleaning, Disinfection, and Sterilization. In: *APIC Infection Control and Applied Epidemiology: Principles and Practice*. Olmstead RN, ed. St. Louis MO; Mosby-YearBook; 1996:

p. 15.1-15.17.

929. Walter CW, Kundsinn RB. The floor as a reservoir of hospital infection. *Surg Gynec Obstet* 1960; 111: 412-416.
930. Scott E, Bloomfield SF. The survival and transfer of microbial contamination via cloths, hands and utensils. *J Appl Bacteriol* 1990; 68: 271-278
931. Scott E, Bloomfield SF. Investigations of the effectiveness of detergent washing, drying and chemical disinfection on contamination of cleaning cloths. *J Appl Bacteriol* 1990; 68: 279-283.
932. Stover B. Personal communication.
933. Givan KF, Black BL, Williams PF. Multiplication of *Pseudomonas* species in phenolic germicidal detergent solution. *Can J Pub Health* 1971; 62: 72.
934. Thomas MEM, Piper E, Maurer IM. Contamination of an operating theater by gram-negative bacteria. Examination of water supplies, cleaning methods, and wound infections. *J Hyg (Camb)* 1972; 70: 63-73.
935. Medcraft JW, Hawkins JM, Fletcher BN, Dadswell JV. Potential hazard from spray cleaning of floors in hospital wards. *J Hosp Infect* 1987; 9: 151-157.
936. Brown DG, Schaltzle K, Gable T. The hospital vacuum cleaner: Mechanism for redistributing microbial contaminants. *J Environ Health* 1980; 42: 192-196.
937. Wysowski DK, Flynt JW, Goldfield M, et al. Epidemic hyperbilirubinemia and use of a phenolic disinfectant detergent. *Pediatrics* 1978; 61: 165-170.
938. Doan HM, Keith L, Shennan AT. Phenol and neonatal jaundice. *Pediatrics* 1979; 64: 324-325.
939. American Academy of Pediatrics, American College of Obstetricians and Gynecologists. Infection control. In: *Guidelines for Perinatal Care*, 4th Ed. Evanston IL, Washington DC; AAP, ACOG; 2000: p. 269-274.
940. Spire B, Montagnier L, Barré-Sinoussi F, Chermann JC. Inactivation of lymphadenopathy associated virus by chemical disinfectants. *Lancet* 1984; 2: 899-901.
941. Martin LS, McDougal JS, Loskoski SL. Disinfection and inactivation of the human T lymphotropic virus type-III/lymphadenopathy-associated virus. *J Infect Dis* 1985; 152: 400-403.
942. Hanson PJ, Gor D, Jeffries DJ, Collins JV. Chemical inactivation of HIV on surfaces. *Brit Med J* 1989; 298: 862-864.
943. Bloomfield SF, Smith-Burchnell CA, Dalglish AG. Evaluation of hypochlorite-releasing disinfectants against the human immunodeficiency virus (HIV). *J Hosp Infect* 1990; 15: 273-278.
944. Druce JD, Jardine D, Locarnini SA, Birch CJ. Susceptibility of HIV to inactivation by disinfectants and ultraviolet light. *J Hosp Infect* 1995; 30: 167-180.
945. Van Bueren J, Simpson RA, Salman H, Farrelly HD, Cookson BD. Inactivation of HIV-1 by chemical disinfectants: Sodium hypochlorite. *Epidemiol Infect* 1995; 115: 567-579.
946. Prince DL, Prince HN, Thraehart O, et al. Methodological approaches to disinfection of human hepatitis viruses. *J Clin Microbiol* 1993; 31: 3296-3304.
947. Tabor E, Gerety RJ. A survey of formalin inactivation of hepatitis A virus, hepatitis B virus, and a non-A, non-B hepatitis agent. In: *Second International Max von Pettenkofer Symposium on Viral Hepatitis*. Munich Germany; October 1982.
948. Thraehart O, Kuwert EK, Scheiermann N, et al. Comparison of the morphological alteration and disintegration test (MADT) and the chimpanzee infectivity test for determination of hepatitis B virucidal activity of chemical disinfectants. *Zentralbl Bakteriol Mikrobiol Hyg (B)* 1982; 176: 472-484.
949. Centers for Disease Control. Recommendations for prevention of HIV transmission in health-care settings. *MMWR* 1987; 36 (No. 2S): 1S-18S.
950. Weber DJ, Barbee SL, Sobsey MD, Rutala WA. The effect of blood on the antiviral activity of sodium hypochlorite, a phenolic, and a quaternary ammonium compound. *Infect Control Hosp Epidemiol* 1999; 20: 821-827.
951. Sattar SA, Springthorpe VS. Survival and disinfectant inactivation of the human immunodeficiency virus: A critical review. *Rev Infect Dis* 1991; 13: 430-437.
952. Centers for Disease Control and Prevention (CDC), National Institutes of Health (NIH). *Biosafety in Microbiological and Biomedical Laboratories*, 4th Ed. Washington DC; U.S. Government Printing Office; 1999.
953. Lee R. The advantages of carpets in mental hospitals. *Ment Hosp* 1965; 16: 324-325.
954. Simmons D, Reizenstein J, Grant M. Considering carpets in hospital use. *Dimensions* 1982; June: 18-21.
955. Willmott M. The effect of a vinyl floor surface and a carpeted floor surface upon walking in elderly hospital in-patients. *Age Ageing* 1986; 15: 119-120.
956. Shaffer J, Key I. The microbiological effects of carpeting on the hospital environment. *Hospitals JAHA* 1966; 40: 126-139.

957. Walter W, Stober A. Quantitative and qualitative microbial studies of hospital carpets. *J Environ Health* 1967; 30: 293-300.
958. Anderson RL. Biological evaluation of carpeting. *Appl Microbiol* 1969; 18: 180-187.
959. Lanese RR, Keller MD, Macpherson CR, Covey RC. A study of microflora on tiled and carpeted surfaces in a hospital nursery. *Am J Public Health* 1973; 63: 174-178.
960. Bonde GJ. Bacterial flora of synthetic carpets in hospitals. *Health Lab Sci* 1973; 10: 308-318.
961. Rylander R, Myrback K, Verner-Carlson B, Ohrstrom M. Bacteriological investigation of wall-to-wall carpeting. *Am J Public Health* 1974; 64: 163-168.
962. Suzuki A, Namba Y, Matsuura M, Horisawa A. Bacterial contamination of floors and other surfaces in operating rooms: A five-year survey. *J Hyg (Camb)* 1984; 93: 559-566.
963. Skoutelis AT, Westenfelder GO, Beckerdite M, Phair JP. Hospital carpeting and epidemiology of *Clostridium difficile*. *Am J Infect Control* 1993; 22: 212-217.
964. Anderson RL, Mackel DC, Stoler BS, Mallison GF. Carpeting in hospitals: An epidemiological evaluation. *J Clin Microbiol* 1982; 15: 408-415.
965. Bakker PGH, Faoagali JL. The effect of carpet on the number of microbes in the hospital environment. *N Zeal Med J* 1977; 85: 88-92.
966. Richet H, McNeil M, Pewters W, et al. *Aspergillus flavus* in a bone marrow transplant unit (BMTU): Pseudofungemia traced to hallway carpeting. *Abstracts of the 89th Annual Meeting of the American Society for Microbiology*; 1989; Abstract F-23, p. 462.
967. Centers for Disease Control. Respiratory illness associated with carpet cleaning at a hospital clinic - Virginia. *MMWR* 1983; 32: 378, 383-384.
968. Maley MP. Bacterial threats to new hospitals. *Lancet* 1997; 350: 223-224.
969. U.S. Department of Labor, Occupational Safety and Health Administration. OSHA Standards Interpretation and Compliance Letters; 6/10/94: Decontamination of a plush carpet surface after a spill. http://www.osha-slc.gov/OshDoc/Interp_data/I19940610.html
970. Richards K. New York City. Special suites, Memorial Sloan Kettering. *Interiors* 1998; 157: 56-59.
971. Noskin BA, Bednarz P, Suriano T, Reiner S, Peterson LR. Persistent contamination of fabric-covered furniture by vancomycin-resistant enterococci: Implications for upholstery selection in hospitals. *Am J Infect Control* 2000; 28: 311-313.
972. Sanderson PJ, Alshafi KM. Environmental contamination by organisms causing urinary tract infection. *J Hosp Infect* 1995; 29: 301-303.
973. Babe KS Jr, Arlian LG, Confer PD, Kim R. House dust mite (*Dermatophagoides farinae* and *Dermatophagoides pteronyssinus*) prevalence in the rooms and hallways of a tertiary care hospital. *J Allergy Clin Immunol* 1995; 95: 801-805.
974. Custovic A, Fletcher A, Pickering CAC, et al. Domestic allergens in public places III: House dust mite, cat, dog, and cockroach allergens in British hospitals. *Clin Exper Allergy* 1998; 28: 53-59.
975. Ansorg R, Thomssen R, Stubbe P. [*Erwinia* species causing fatal septicemia in a newborn.] *Med Microbiol Immunol (Berl)* 1974; 159: 161-170. (German)
976. Trust TJ, Bartlett KH. Isolation of *Pseudomonas aeruginosa* and other bacterial species from ornamental aquarium plants. *Appl Environ Microbiol* 1976; 31: 992-994.
977. Bartzokas CA, Holley MP, Sharp CA. Bacteria in flower vase water: incidence and significance in general ward practice. *Br J Surg* 1975; 62: 295-297.
978. Watson AG, Koons CE. *Pseudomonas* on the chrysanthemums. *Lancet* 1973; 2: 91.
979. Siegman-Igra Y, Shalem A, Berger SA, Livio S, Michaeli D. Should potted plants be removed from hospital wards? *J Hosp Infect* 1986; 7: 82-85.
980. Rosenzweig AL. Contaminated flower vases. *Lancet* 1973; 2: 568-569.
981. Johansen KS, Laursen H, Wilhjelm BJ. Flower vases as reservoirs of pathogens. *Lancet* 1974; 1: 359.
982. Rogues AM, Quesnel C, Revel P, Saric J, Gachie JP. Potted plants as a potential reservoir of *Fusarium* species. *J Hosp Infect* 1997; 35: 163-164.
983. Levine OS, Levine MM. Houseflies (*Musca domestica*) as mechanical vectors of shigellosis. *Rev Infect Dis* 1991; 13: 688-696.
984. Šrámová H, Daniel M, Absolonová V, D•di..ová D, Jedli..ková Z, Lhotová H, Petráš P, Subertová V. Epidemiological role of arthropods detectable in health facilities. *J Hosp Infect* 1992; 20: 281-292.
985. Tan SW, Yap KL, Lee HL. Mechanical transport of rotavirus by the legs and wings of *Musca domestica*

(Diptera: *Muscidae*). *J Med Entomol* 1997; 34: 527-531.

986. Burgess NRH. Hospital design and cockroach control. *Trans R Soc Trop Med Hyg* 1984; 78: 293-294.
987. Ash N, Greenberg B. Vector potential for the German cockroach (Diptera: *Blattellidae*) in dissemination of *Salmonella enteritidis* serotype typhimurium. *J Med Entomol* 1980; 17: 417-423.
988. Fotedar R, Banerjee U. Nosocomial fungal infections - Study of the possible role of cockroaches (*Blattella germanica*) as vectors. *Acta Trop* 1992; 50: 339-343.
989. Rosef O, Kapperud G. House flies (*Musca domestica*) as possible vectors of *Campylobacter fetus* subsp. *jejuni*. *Appl Environ Microbiol* 1983; 45: 381-383.
990. Forsey T, Darougar S. Transmission of chlamydiae by the housefly. *Br J Ophthalmol* 1981; 65: 147-150.
991. Grübel P, Hoffman JS, Chong FK, Burstein NA, Mepani C, Cave DR. Vector potential of houseflies (*Musca domestica*) for *Helicobacter pylori*. *J Clin Microbiol* 1997; 35: 1300-1303.
992. Oothuman P, Jeffery J, Aziz, AHA, Bakar EA, Jegathesan M. Bacterial pathogens isolated from cockroaches trapped from pediatric wards in peninsular Malaysia. *Trans R Soc Trop Med Hyg* 1989; 83: 133-135.
993. Beatson SH. Pharaoh's ants as pathogen vectors in hospitals. *Lancet* 1972; 1: 425-427.
994. LeGuyader A, Rivault C, Chaperon J. Microbial organisms carried by brown-banded cockroaches in relation to their spatial distribution in a hospital. *Epidemiol Infect* 1989; 102: 485-492.
995. Fotedar R, Banerjee U, Shriniwas, Verma A. Cockroaches (*Blattella germanica*) as carriers of microorganisms of medical importance in hospitals. *Epidemiol Infect* 1991; 107: 181-187.
996. Fotedar R, Banerjee U, Singh S, Shriniwas, Verma AK. The housefly (*Musca domestica*) as a carrier of pathogenic microorganisms in a hospital environment. *J Hosp Infect* 1992; 20: 209-215.
997. Fotedar R, Shriniwas, Banerjee U, Samantray JC, Nayar E, Verma A. Nosocomial infections: cockroaches as possible vectors of drug-resistant *Klebsiella*. *J Hosp Infect* 1991; 18: 155-159.
998. Devi SJN, Murray CJ. Cockroaches (*Blatta* and *Periplaneta* species) as reservoirs of drug-resistant salmonellas. *Epidemiol Infect* 1991; 107: 357-364.
999. Baker LF. Pests in hospitals. *J Hosp Infect* 1981; 2:5-9.
1000. Allen BW. Excretion of viable tubercle bacilli by *Blatta orientalis* (the oriental cockroach) following ingestion of heat-fixed sputum smears: A laboratory investigation. *Trans R Soc Trop Med Hyg* 1987; 81: 98-99.
1001. Technical Guide. Sputum examination for tuberculosis by direct microscopy. *Bulletin of the International Union Against Tuberculosis*, Supplement No. 2.
1002. Cohen D, Green M, Block C, Slepon R, Ambar R, Wasserman SS, Levine MM. Reduction of transmission of shigellosis by control of houseflies (*Musca domestica*). *Lancet* 1991; 337: 993-997.
1003. Watkins M, Wyatt T. A ticklish problem: Pest infestation in hospitals. *Prof Nurse* 1989; 4: 389-392.
1004. Schoninger S. Pest control and extermination in health care facilities. *Prof Sanit Manage* 1978; 9: 24-27.
1005. Bruesch J. Institutional pest management: Current trends. *Exec Housekeep Today* 1994; 15: 6-12.
1006. Tenover FC. VRSA, VISA, GISA: The dilemma behind the name game. *Clin Microbiol Newsletter* 2000; 22: 49-53.
1007. Centers for Disease Control and Prevention. National Nosocomial Infections Surveillance System (NNIS); Semiannual report; December 1999. <http://www.cdc.gov/ncidod/hip/SURVEILL/NNIS.HTM>
1008. Hartstein AI, Mulligan ME. Methicillin-resistant *Staphylococcus aureus*. In: *Hospital Epidemiology and Infection Control*, 2nd Ed. Mayhall CG, ed. Philadelphia PA; Lippincott Williams & Wilkins; 1999: p. 347-364.
1009. Walsh TJ, Vlahov D, Hansen SL, et al. Prospective microbiologic surveillance in control of nosocomial methicillin-resistant *Staphylococcus aureus*. *Infect Control* 1987;8: 7-14.
1010. Walsh TJ, Auger F, Tatem BA, Hansen SL, Standford HJ. Novobiocin and rifampin in combination against methicillin-resistant *Staphylococcus aureus*: An *in vitro* comparison with vancomycin plus rifampin. *J Antimicrob Chemother* 1986; 17: 75-82.
1011. McNeil MM, Solomon SL. The epidemiology of MRSA. *Antimicrobiol Newsl* 1985; 2: 49-56.
1012. Oie S, Kamiya A. Survival of methicillin-resistant *Staphylococcus aureus* (MRSA) on naturally contaminated dry mops. *J Hosp Infect* 1996; 34: 145-149.
1013. Arnow PM, Allyn PA, Nichols EM, Hill DL, Pezzlo M, Bartlett RH. Control of methicillin-resistant *Staphylococcus aureus* in a burn unit: Role of nurse staffing. *J Trauma* 1982; 22: 954-959.
1014. Karanfil LV, Murphy M, Josephson A, et al. A cluster of vancomycin-resistant *Enterococcus faecium* in an intensive care unit. *Infect Control Hosp Epidemiol* 1992; 13: 195-200.
1015. Handwerger S, Raucher B, Altarac D, et al. Nosocomial outbreak due to *Enterococcus faecium* highly resistant to vancomycin, penicillin, and gentamicin. *Clin Infect Dis* 1993; 16: 750-755.

1016. Boyle JF, Soumakis SA, Rendo A, et al. Epidemiologic analysis and genotypic characterization of a nosocomial outbreak of vancomycin-resistant enterococci. *J Clin Microbiol* 1993; 31: 1280-1285.
1017. Boyce JM, Opal SM, Chow JW, et al. Outbreak of multidrug-resistant *Enterococcus faecium* with transferable *vanB* class vancomycin resistance. *J Clin Microbiol* 1994; 32: 1148-1153.
1018. Rhinehart E, Smith NE, Wennerstein C, et al. Rapid dissemination of beta-lactamase-producing, aminoglycoside-resistant *Enterococcus faecalis* among patients and staff on an infant-toddler surgical ward. *N Engl J Med* 1990; 323: 1814-1818.
1019. Crossley K, Landesman B, Zaske D. An outbreak of infections caused by strains of *Staphylococcus aureus* resistant to methicillin and aminoglycosides. II. Epidemiologic studies. *J Infect Dis* 1979; 139: 280-287.
1020. Peacock JE Jr, Marsik FJ, Wenzel RP. Methicillin-resistant *Staphylococcus aureus*: Introduction and spread within a hospital. *Ann Intern Med* 1980; 93: 526-532.
1021. Walsh TJ, Hansen SL, Tatem BA, Auger F, Standiford HJ. Activity of novobiocin against methicillin-resistant *Staphylococcus aureus*. *J Antimicrob Chemother* 1985; 15: 435-440.
1022. Livornese LL Jr, Sias S, Samel C, et al. Hospital-acquired infection with vancomycin-resistant *Enterococcus faecium* transmitted by electronic thermometers. *Ann Intern Med* 1992; 117: 112-116.
1023. Gould FK, Freeman R. Nosocomial infection with microsphere beds. *Lancet* 1993; 342: 241-242.
1024. Morris JG, Shay DK, Hebden JN, et al. Enterococci resistant to multiple antimicrobial agents, including vancomycin: Establishment of endemicity in a university medical center. *Ann Intern Med* 1995; 123: 250-259.
1025. Edmond MB, Ober JS, Weinbaum DL, et al. Vancomycin-resistant *Enterococcus faecium* bacteremia: Risk factors for infection. *Clin Infect Dis* 1995; 20: 1126-1133.
1026. Zervos MJ, Kauffman CA, Therasse PM, Bergman AG, Mikesell TS, Schaberg DR. Nosocomial infection by gentamicin-resistant *Streptococcus faecalis*: An epidemiologic study. *Ann Intern Med* 1987; 106: 687-691.
1027. Zervos MJ, Dembinski S, Mikesell T, Schaberg DR. High-level resistance to gentamicin in *Streptococcus faecalis*: Risk factors and evidence for exogenous acquisition of infection. *J Infect Dis* 1986; 153: 1075-1083.
1028. Bonilla HF, Zervos MA, Lyons MJ, et al. Colonization with vancomycin-resistant *Enterococcus faecium*: Comparison of a long-term-care unit with an acute-care hospital. *Infect Control Hosp Epidemiol* 1997; 18: 333-339.
1029. Bonilla HF, Zervos MJ, Kauffman CA. Long-term survival of vancomycin-resistant *Enterococcus faecium* on a contaminated surface. *Infect Control Hosp Epidemiol* 1996; 17: 770-771
1030. Boyce JM, Bermel LA, Zervos MJ, et al. Controlling vancomycin-resistant enterococci. *Infect Control Hosp Epidemiol* 1995; 16: 634-637.
1031. Bonten MJM, Hayden MK, Nathan C, et al. Epidemiology of colonisation of patients and environmental with vancomycin-resistant enterococci. *Lancet* 1996; 348: 1615-1619.
1032. Wendt C, Wiesenthal B, Dietz E, Rüden H. Survival of vancomycin-resistant and vancomycin-susceptible enterococci on dry surfaces. *J Clin Microbiol* 1998; 36: 3734-3746.
1033. Bradley CR, Fraiese AP. Heat and chemical resistance of enterococci. *J Hosp Infect* 1996; 34: 191-196.
1034. Anderson RL, Carr JH, Bond WW, Favero MS. Susceptibility of vancomycin-resistant enterococci to environmental disinfectants. *Infect Control Hosp Epidemiol* 1997; 18: 195-199.
1035. Saurina G, Landman D, Quale JM. Activity of disinfectants against vancomycin-resistant *Enterococcus faecium*. *Infect Control Hosp Epidemiol* 1997; 18: 345-347.
1036. Rutala WA, Stiegel MM, Sarubbi FA, Weber DJ. Susceptibility of antibiotic-susceptible and antibiotic-resistant hospital bacteria to disinfectants. *Infect Control Hosp Epidemiol* 1997; 18: 417-421.
1037. Schulster LM, Anderson RL. Susceptibility of glycopeptide-intermediate resistant *Staphylococcus aureus* (GISA) to surface disinfectants, hand washing chemicals, and a skin antiseptic. In: *Abstracts of the 98th General Meeting, American Society for Microbiology*. 1998; Abstract Y-3: p. 547.
1038. Armstrong-Evans M, Litt M, McArthur MA, et al. Control of transmission of vancomycin-resistant *Enterococcus faecium* in a long-term-care facility. *Infect Control Hosp Epidemiol* 1999; 20: 312-317.
1039. Freeman R, Kearns AM, Lightfoot NF. Heat resistance of nosocomial enterococci. *Lancet* 1994; 345: 64-65.
1040. Weber DJ, Rutala WA. Role of environmental contamination in the transmission of vancomycin-resistant enterococci. *Infect Control Hosp Epidemiol* 1997; 18:306-309.
1041. Lai KK, Kelley AL, Melvin ZS, Belliveau PP, Fontecchio SA. Failure to eradicate vancomycin-resistant enterococci in a university hospital and the cost of barrier precautions. *Infect Control Hosp Epidemiol* 1998; 19: 647-652.
1042. Byers KE, Durbin LJ, Simonton BM, Anglim AM, Adal KA, Farr BM. Disinfection of hospital rooms contaminated with vancomycin-resistant *Enterococcus faecium*. *Infect Control Hosp Epidemiol* 1998; 19: 261-264.

1043. Siegel DL, Edelstein PH, Nachamkin I. Inappropriate testing for diarrheal diseases in the hospital. *JAMA* 1990; 263: 979-982.
1044. Yannelli B, Gurevich I, Schoch PE, Cunha BA. Yield of stool cultures, ova and parasite tests, and *Clostridium difficile* determination in nosocomial diarrhea. *Am J Infect Control* 1988; 16: 246-249.
1045. Gerding DN, Olson MM, Peterson LR, et al. *Clostridium difficile*-associated diarrhea and colitis in adults: A prospective case-controlled epidemiologic study. *Arch Intern Med* 1986; 146: 95-100.
1046. Barlett JG. Antibiotic-associated colitis. *Dis Mon* 1984; 30: 1-55.
1047. Pierce PF Jr, Wilson R, Silva J Jr, et al. Antibiotic-associated pseudomembranous colitis: An epidemiologic investigation of a cluster of cases. *J Infect Dis* 1982; 145: 269-274.
1048. Aronsson B, Möllby, Nord C-E. Antimicrobial agents and *Clostridium difficile* in acute enteric disease: Epidemiologic data from Sweden, 1980-1982. *J Infect Dis* 1985; 151: 476-481.
1049. Thibault A, Miller MA, Gaese C. Risk factors for the development of *Clostridium difficile*-associated diarrhea during a hospital outbreak. *Infect Control Hosp Epidemiol* 1991; 12: 345-348.
1050. McFarland LV, Surawicz CM, Stamm WE. Risk factors for *Clostridium difficile* carriage and *Clostridium difficile*-associated diarrhea in a cohort of hospitalized patients. *J Infect Dis* 1990; 162: 678-684.
1051. Zadik PM, Moore AP. Antimicrobial associations of an outbreak of diarrhoea due to *Clostridium difficile*. *J Hosp Infect* 1998; 39: 189-193.
1052. Johnson S, Homann SR, Bettin KM, et al. Treatment of asymptomatic *Clostridium difficile* carriers (fecal excretors) with vancomycin or metronidazole. A randomized, placebo controlled trial. *Ann Intern Med* 1992; 117: 297-302.
1053. Gerding DN, Johnson S, Peterson LR, Mulligan ME, Silva J Jr. *Clostridium difficile*-associated diarrhea and colitis. *Infect Control Hosp Epidemiol* 1995; 16: 459-477.
1054. Titov L, Lebedkova N, Shabanov A, Tang YJ, Cohen SH, Silva J Jr. Isolation and molecular characterization of *Clostridium difficile* strains from patients and the hospital environment in Belarus. *J Clin Microbiol* 2000; 38: 1200-1202.
1055. Mulligan ME, Rolfe RD, Finegold SM, George WL. Contamination of a hospital environment by *Clostridium difficile*. *Curr Microbiol* 1979; 3: 173-175.
1056. Fekety R, Kim KH, Brown D, Batts DH, Cudmore M, Silva J Jr. Epidemiology of antibiotic-associated colitis: Isolation of *Clostridium difficile* from the hospital environment. *Am J Med* 1981; 70: 906-908.
1057. Malamou-Ladas H, Farrell SO, Nash JO, Tabaqchali S. Isolation of *Clostridium difficile* from patients and the environment of hospital wards. *J Clin Pathol* 1983; 6: 88-92.
1058. Kaatz GW, Gitlin SD, Schaberg DR, et al. Acquisition of *Clostridium difficile* from the hospital environment. *Am J Epidemiol* 1988; 127: 1289-1294.
1059. Cohen SH, Tang YJ, Muenzer J, Gumerlock PH, Silva J Jr. Isolation of various genotypes of *Clostridium difficile* from patients and the environment in an oncology ward. *J Infect Dis* 1997; 889-893.
1060. Savage AM. Nosocomial spread of *Clostridium difficile*. *Infect Control* 1983; 4: 31-33.
1061. Brooks SE, Veal RO, Kramer M, Dore L, Schupf N, Adachi M. Reduction in the incidence of *Clostridium difficile*-associated diarrhea in an acute care hospital and a skilled nursing facility following replacement of electronic thermometers with single-use disposables. *Infect Control Hosp Epidemiol* 1992; 13: 98-103.
1062. Johnson S, Gerding DN, Olson MM, et al. Prospective, controlled study of vinyl glove use to interrupt *Clostridium difficile* nosocomial transmission. *Am J Med* 1990; 88: 137-140.
1063. Worsley MA. Infection control and prevention of *Clostridium difficile* infection. *J Antimicrobial Chemother* 1998; 41 (Suppl. C): 59-66.
1064. Hall CB, Douglas G Jr, Gelman JM. Possible transmission by fomites of respiratory syncytial virus. *J Infect Dis* 1980; 141: 98-102.
1065. Brady MT, Evans J, Cuartas J. Survival and disinfection of parainfluenza viruses on environmental surfaces. *Am J Infect Control* 1990; 18: 18-23.
1066. Hendley JO, Wenzel RP, Gwaltney JM Jr. Transmission of rhinovirus colds by self-inoculation. *N Engl J Med* 1973; 288: 1361-1364.
1067. Butz AM, Fosarelli P, Dick J, Cusack T, Yolken R. Prevalence of rotavirus on high-risk fomites in day-care facilities. *Pediatrics* 1993; 92: 202-205.
1068. Wilde J, Van R, Pickering LK, Eiden J, Yolken R. Detection of rotaviruses in the day care environment - Detection by reverse transcriptase polymerase chain reaction. *J Infect Dis* 1992; 166: 507-511.
1069. Chapin M, Yatabe J, Cherry JD. An outbreak of rotavirus gastroenteritis on a pediatric unit. *Am J Infect*

Control 1983; 11: 88-91.

1070. Chadwick PR, Beards G, Brown D, et al. Management of hospital outbreaks of gastro-enteritis due to small round structured viruses. Report of the Public Health Laboratory Service, Viral Gastroenteritis Working Group. *J Hosp Infect* 2000; 45: 1-10.

1071. Spender QW, Lewis D, Price EH. Norwalk-like viruses: Study of an outbreak. *Arch Dis Child* 1986; 61: 142-147.

1072. Storr J, Rice S, Phillips AD, Price E, Walker Smith JA. Clinical associations of Norwalk-like virus in the stools of children. *J Pediatr Gastroenterol Nutr* 1986; 5: 576-580.

1073. Russo PL, Spelman DW, Harrington GA, et al. Hospital outbreak of Norwalk-like virus. *Infect Control Hosp Epidemiol* 1997; 17: 1374-1378.

1074. Springthorpe VS, Grenier JL, Lloyd-Evans N, Sattar SA. Chemical disinfection of human rotaviruses: Efficacy of commercially-available products in suspension tests. *J Hyg (Camb)* 1986; 97: 139-161.

1075. Lloyd-Evans N, Springthorpe VS, Sattar SA. Chemical disinfection of human rotavirus-contaminated inanimate surfaces. *J Hyg (Camb)* 1986; 97: 163-173.

1076. Brown P, Gajdusek DC. The human spongiform encephalopathies: Kuru, Creutzfeldt-Jakob disease, and Gerstmann-Sträussler-Scheinker syndrome. *Curr Top Microbiol Immunol* 1991; 172: 1-20.

1077. Will RG. Epidemiology of Creutzfeldt-Jakob disease. *Brit Med Bull* 1993; 49: 960-970.

1078. Holman RC, Khan AS, Belay ED, Schonberger LB. Creutzfeldt-Jakob disease in the United States, 1979-1994: Using national mortality data to assess the possible occurrence of variant cases. *Emerg Infect Dis* 1996; 2: 333-337.

1079. Will RG, Ironside JW, Zeidler M, et al. A new variant of Creutzfeldt-Jakob disease in the U.K. *Lancet* 1996; 347: 921-925.

1080. Lasmézas CI, Deslys JP, Demaimay R, et al. BSE transmission to macaques. *Nature* 1996; 381: 743-744.

1081. Collinge J, Sidle KCL, Heads J, Ironside J, Hill AF. Molecular analysis of prion strain variation and the aetiology of "new variant" CJD. *Nature* 1996; 383: 685-690.

1082. Bruce ME, Will RG, Ironside JW, et al. Transmission to mice indicates that "new variant" CJD is caused by the BSE agent. *Nature* 1997; 389: 498-501.

1083. Prusiner SB. Biology and genetics of prion diseases. *Ann Rev Microbiol* 1994; 48: 655-686.

1084. Prusiner SB. Human prion diseases. In: *Principles and Practice of Clinical Virology*, 3rd Ed. Zuckerman AJ, Banatvala JE, Pattison JR, eds. Chichester UK; John Wiley & Sons; 1995: p. 703-729.

1085. Prusiner SB. Prions. *Proc Natl Acad Sci USA* 1998; 95: 13363-13383.

1086. Kimberlin RH, Walker CA, Millson GC, et al. Disinfection studies with two strains of mouse-passaged scrapie agent. Guideline for Creutzfeldt-Jakob and related agents. *J Neurol Sci* 1983; 59: 355-349.

1087. Sklaviadis TK, Manuelidis L, Manuelidis EE. Physical properties of the Creutzfeldt-Jakob disease agent. *J Virol* 1989; 63: 1212-1222.

1088. Brown P, Gajdusek DC, Gibbs CJ Jr, Asher DM. Potential epidemic of Creutzfeldt-Jakob disease from human growth hormone therapy. *N Engl J Med* 1985; 12: 728-733.

1089. Brown P, Preece MA, Will RG. "Friendly fire" in medicine: Hormones, homografts and Creutzfeldt-Jakob disease. *Lancet* 1992; 340: 24-27.

1090. Frasier D, Foley TP Jr. Creutzfeldt-Jakob disease in recipients of pituitary hormones. *J Clin Endocrinol Metabol* 1994; 78: 1277-1279.

1091. Centers for Disease Control. Epidemiologic notes and reports: Rapidly progressive dementia in a patient who received a cadaveric dura mater graft. *MMWR* 1987; 36: 49-50, 55.

1092. Centers for Disease Control. Epidemiologic notes and reports update: Creutzfeldt-Jakob disease in a patient receiving cadaveric dura mater graft. *MMWR* 1987; 36: 324-325.

1093. Centers for Disease Control. Epidemiologic notes and reports update: Creutzfeldt-Jakob disease in a second patient who received a cadaveric dura mater graft. *MMWR* 1989; 38: 37-38, 43.

1094. Centers for Disease Control and Prevention. Creutzfeldt-Jakob disease in patients who received a cadaveric dura mater graft - Spain, 1985-1992. *MMWR* 1993; 42: 560-563.

1095. Martinez-Lage JF, Poza M, Sola J, et al. Accidental transmission of Creutzfeldt-Jakob disease by dural cadaveric grafts. *J Neurol Neurosurg Psychiatry* 1994; 57: 1091-1094.

1096. Centers for Disease Control and Prevention. Creutzfeldt-Jakob disease associated with cadaveric dura mater grafts - Japan, January 1979-May 1997. *MMWR* 1997; 46: 1066-1069.

1097. Lang CLG, Heckmann JG, Neundörfer B. Creutzfeldt-Jakob disease via dural and corneal transplants. *J Neurol Sci* 1998; 160: 128-139.

1098. Nevin S, McMenemey WH, Behrman S, Jones DP. Subacute spongiform encephalopathy - A subacute form of encephalopathy attributable to vascular dysfunction (spongiform cerebral atrophy). *Brain* 1960; 83: 519-569.
1099. Bernoulli C, Siegfried J, Baumgartner G, et al. Danger of accidental person-to-person transmission of Creutzfeldt-Jakob disease by surgery. *Lancet* 1977; 1: 478-479.
1100. Will RG, Matthews WB. Evidence for case-to-case transmission of Creutzfeldt-Jakob disease. *J Neurol Neurosurg Psychiatry* 1982; 45: 235-238.
1101. El Hachimi KH, Chaunu M-P, Cervenakova L, Brown P, Foncin J-F. Putative neurosurgical transmission of Creutzfeldt-Jakob disease with analysis of donor and recipient: Agent strains. *Comp Rendus Acad Sci Iii: Science de la vie* 1997; 320: 319-328.
1102. Brown P, Gibbs CJ, Amyx HL, et al. Chemical disinfection of Creutzfeldt-Jakob disease virus. *N Engl J Med* 1982; 306: 1279-1282.
1103. Brown P, Rohwer RG, Gajdusek DC. Newer data on the inactivation of scrapie virus or Creutzfeldt-Jakob disease virus in brain tissue. *J Infect Dis* 1986; 153: 1145-1148.
1104. Rosenberg RN, White CL, Brown P, Gajdusek DC, Volpe JJ, Dyck PJ. Precautions in handling tissues, fluids, and other contaminated materials from patients with documented or suspected Creutzfeldt-Jakob disease. *Ann Neurol* 1986; 19: 75-77.
1105. Taylor DM. Resistance of the ME7 scrapie agent to peracetic acid. *Vet Microbiol* 1991; 27: 19-24.
1106. Taguchi F, Tamai Y, Uchida K, et al. Proposal for a procedure for complete inactivation of the Creutzfeldt-Jakob disease agent. *Arch Virol* 1991; 119: 297-301.
1107. Taylor D. Inactivation of the unconventional agents of scrapie, bovine spongiform encephalopathy, and Creutzfeldt-Jakob disease. *J Hosp Infect* 1991; 18: 141-146.
1108. Favero MS. Current issues in hospital hygiene and sterilization technology. *J Infect Control (Asia Pacific Edition)* 1998; 1: 8-10.
1109. Ricketts MN, Cashman NR, Stratton EE, El Saadany S. Is Creutzfeldt-Jakob disease transmitted in blood? *Emerg Infect Dis* 1997; 3: 155-163.
1110. Will RG, Kimberlin RH. Creutzfeldt-Jakob disease and the risk from blood or blood products. *Vox Sang* 1998; 75: 178-180.
1111. Evatt B, Austin H, Barnhart E, et al. Surveillance for Creutzfeldt-Jakob disease among persons with hemophilia. *Transfusion* 1998; 38: 817-820.
1112. Patry D, Curry B, Easton D, Mastrianni JA, Hogan DB. Creutzfeldt-Jakob disease (CJD) after blood product transfusion from a donor with CJD. *Neurology* 1998; 50: 1872-1873.
1113. Budka H, Aguzzi A, Brown P, et al. Tissue handling in suspected Creutzfeldt-Jakob disease (CJD) and other human spongiform encephalopathies (prion diseases). *Brain Pathol* 1995; 5: 319-322.
1114. Ironside JW, Bell JE. The "high-risk" neuropathological autopsy in AIDS and Creutzfeldt-Jakob disease: Principles and practice. *Neuropathol Appl Neurobiol* 1996; 22: 388-393.
1115. Litsky BY. Results of bacteriological surveys highlight problem areas in hospitals. *Hospital Management* 1966; 101: 82-88.
1116. Eickhoff TC. Microbiologic sampling. *Hospitals* 1970; 44: 86-87.
1117. American Hospital Association Committee on Infections Within the Hospitals. *Statement on microbiologic sampling in the hospital* 1974; 48: 125-126.
1118. Rafferty KM, Pancoast SJ. Brief report: Bacteriological sampling of telephones and other hospital staff-hand contact objects. *Infect Control* 1984; 5: 533-535.
1119. Haley RW, Shachtman RS. The emergence of infection control programs in U.S. hospitals: An assessment, 1976. *Am J Epidemiol* 1980; 111: 574-591.
1120. Mallison GF, Haley RW. Microbiologic sampling of the inanimate environment in U.S. hospitals, 1976-1977. *Am J Med* 1981; 70: 941-946.
1121. Gröschel DHM. Air sampling in hospitals. *Ann NY Acad Sci* 1980; 353: 230-240.
1122. Barbaree JM, Gorman GW, Martin WT, Fields BS, Morrill WE. Protocol for sampling environmental sites for legionellae. *Appl Environ Microbiol* 1987; 53: 1454-1458.
1123. Eickhoff TC. Microbiologic sampling of the hospital environment. *Health Lab Sci* 1974; 11: 73-75.
1124. Isenberg HD. Significance of environmental microbiology in nosocomial infections and the care of hospitalized patients. In: *Significance of Medical Microbiology in the Care of Patients*; Lorian V, ed. Baltimore MD; Williams & Wilkins; 1977: p. 220-234.
1125. McGowan JE Jr, Weinstein RA. The role of the laboratory in control of nosocomial infection. In: *Hospital*

Infections, 4th Ed; Bennett JV, Brachman PS, eds. Philadelphia PA; Lippincott Raven: 1998; p. 143-164.

1126. Turner AG, Wilkins JR, Craddock JG. Bacterial aerosolization from an ultrasonic cleaner. *J Clin Microbiol* 1975; 1: 289-293.

1127. Bond WW, Hedrick ER. Microbiological culturing of environmental and medical-device surfaces. In: *Clinical Microbiology Procedures Handbook, Section 11*; Isenberg HD, Gilchrist MJR, eds. Washington DC; American Society for Microbiology Press; 1992: p. 11.10.1-11.10.9.

1128. Cole EC, Cook CE. Characterization of infectious aerosols in health care facilities: An aid to effective engineering controls and preventive strategies. *Am J Infect Control* 1998; 26: 452-464.

1129. Nevalainen A, Willeke K, Liebhaber F, Pastuszka J, Burge H, Henningson E. Bioaerosol sampling. In: *Aerosol Management*; Willeke K, Baron PA, eds. New York NY; Van Nostrand Reinhold; 1993: p. 471-492.

1130. Cox CS. The aerobiological pathway of microorganisms. Chichester UK; John Wiley & Sons; 1987.

1131. Wolf HW, Skaliy P, Hall LB, et al. Sampling microbiological aerosols. Public Health Service publication No. 686. Government Printing Office, Washington, DC; 1964.

1132. Zeterberg JM. A review of respiratory virology and the spread of virulent and possible antigenic viruses via air conditioning systems. *Ann Allergy* 1973; 31: 228-234.

1133. Randall CW, Ledbetter JO. Bacterial air pollution from activated sludge units. *Am Ind Hyg Assoc J* 1966; Nov/Dec: 506-519.

1134. Salem H, Gardner DE. Health aspects of bioaerosols. In: *Atmospheric Microbial Aerosols, Theory and Applications*, Lighthart B, Mohr AJ, eds. New York NY; Chapman and Hall; 1985: p. 304-330.

1135. Sattar SA, Ijaz MK. Spread of viral infections by aerosols. *Crit Rev Environ Control* 1987; 17: 89-131.

1136. Buttner MP, Willeke K, Grinshpun SA. Sampling and analysis of airborne microorganisms. In: *Manual of Environmental Microbiology*; Hurst CJ, Knudsen GR, McInerney MJ, Stetzenbach LD, Walter MV, eds. Washington DC; American Society for Microbiology Press; 1997: p. 629-640.

1137. Jensen PA, Schafer MP. Sampling and characterization of bioaerosols. In: *NIOSH Manual of Analytical Methods*; Cincinnati OH; Centers for Disease Control and Prevention; 1998: p. 82-112.

<http://www.cdc.gov/niosh/nmam/pdfs/chapter-j.pdf>

1138. Jolley AE. The value of surveillance cultures on neonatal intensive care units. *J Hosp Infect* 1993; 25: 153-159.

1139. Hardy KA, McGowan KL, Fisher MC, Schidlow DV. *Pseudomonas cepacia* in the hospital setting: Lack of transmission between cystic fibrosis patients. *J Pediatr* 1986; 109: 51-54.

1140. Hambræus A, Lagerqvist-Widh A, Zettersten U, Engberg S, Sedin G, Sjöberg L. Spread of *Klebsiella* in a neonatal ward. *Scand J Infect Dis* 1991; 23: 189-194.

1141. Humphreys H, Peckham D, Patel P, Knox A. Airborne dissemination of *Burkholderia (Pseudomonas) cepacia* from adult patients with cystic fibrosis. *Thorax* 1994; 49: 1157-1159.

1142. Pankhurst CL, Harrison VE, Philpott-Howard J. Evaluation of contamination of the dentist and dental surgery environment with *Burkholderia (Pseudomonas) cepacia* during treatment of children with cystic fibrosis. *Int J Paediatr Dent* 1995; 5: 243-247.

1143. Favero MS, Bond WW. Chemical disinfection of medical and surgical materials. In: *Disinfection, Sterilization, and Preservation*, 5th Ed; Block SS. ed. Philadelphia PA; Lippincott Williams & Wilkins; 2000: p. 881-917.

1144. Weber DO, Gooch JJ, Wood WR, Britt EM, Kraft RO. Influence of operating room surface contamination on surgical wounds: A prospective study. *Arch Surg* 1976; 111: 484-488.

1145. Pfeiffer EH, Wittig JR, Dunkelberg H, Werner HP. Hygienic and bacteriological comparative studies in 50 hospitals. V. Bacterial contamination of hospital surfaces. *Zentralbl Bakteriol [B]* 1978; 167: 11-21. (German)

1146. Sattar SA, Lloyd-Evans N, Springthorpe VS. Institutional outbreaks of rotavirus diarrhea: Potential role of fomites and environmental surfaces as vehicles for virus transmission. *J Hyg (Camb)* 1986; 96: 277-289.

1147. Smith SM, Eng RH, Padberg FT Jr. Survival of nosocomial pathogenic bacteria at ambient temperature. *J Med* 1996; 27: 293-302.

1148. Craythorn JM, Barbour AG, Matsen JM, Britt MR, Garibaldi RA. Membrane filter contact technique for bacteriological sampling of moist surfaces. *J Clin Microbiol* 1980; 12: 250-255.

1149. Scott E, Bloomfield SF, Barlow CG. A comparison of contact plate and calcium alginate swab techniques for quantitative assessment of bacteriological contamination of environmental surfaces. *J Appl Bacteriol* 1984; 56: 317-320.

1150. Poletti L, Pasquarella C, Pitzurra M, Savino A. Comparative efficiency of nitrocellulose membranes versus RODAC plates in microbial sampling on surfaces. *J Hosp Infect* 1999; 41: 195-201.

1151. International Organization for Standardization (ISO). Sterilization of medical devices - Microbiological methods, Part 1. ISO Standard 11737-1. Paramus NJ; International Organization for Standardization; 1995.

1152. Russell AD. Factors influencing the efficacy of antimicrobial agents. In: *Principles and Practices of Disinfection, Preservation and Sterilization*, Russell AD, Hugo WB, Ayliffe GAJ, eds. Oxford UK; Blackwell Science; 1999: p. 95-123.
1153. Favero MS, Gabis DA, Vesley D. Environmental monitoring procedures. In: *Compendium of Methods for the Microbiological Examination of Foods*, 2nd Ed, Speck ML ed. Washington DC; American Public Health Association; 1984: p. 47-61.
1154. Favero MS, Bond WW, Petersen NJ, Berquist KR, Maynard JE. Detection methods for study of the stability of hepatitis B antigen on surfaces. *J Infect Dis* 1974; 129: 210-212.
1155. Favero MS, McDade JJ, Robertsen JA, Hoffmann RK, Edwards RW. Microbiological sampling of surfaces. *J Appl Bacteriol* 1968; 31: 336-343.
1156. Petersen NJ, Collins DE, Marshall JH. Evaluation of skin cleansing procedures using the wipe-rinse technique. *Health Lab Sci* 1974; 11: 182-197.
1157. Schalkowsky S, Hall LB, Kline RC. Potential effects of recent findings on spacecraft sterilization requirements. *Space Life Sci* 1969; 1: 520-530.
1158. Hall LB, Lyle RG. Foundations of planetary quarantine. *Environ Biol Med* 1971; 1: 5-8.
1159. Rutala WA, Weber DJ. Uses of inorganic hypochlorite (bleach) in health-care facilities. *Clin Microbiol Rev* 1997; 10: 597-610.
1160. Mallison GF. Central services and linens and laundry. In: *Hospital Infections*, Bennett JV, Brachman PS, eds. Boston MA; Little, Brown, & Co; 1986: p. 251-256.
1161. Blaser MJ, Smith PE, Cody HJ, Wang W-LL, LaForce FM. Killing of fabric-associated bacteria in hospital laundry by low-temperature washing. *J Infect Dis* 1984; 149: 48-57.
1162. Centers for Disease Control. Outbreak of viral hepatitis in the staff of a pediatric ward - California. *MMWR* 1977; 28: 77-79.
1163. Shah PC, Krajden S, Kane J, Summerbell RC. Tinea corporis caused by *Microsporum canis*: Report of a nosocomial outbreak. *Eur J Epidemiol* 1988; 4: 33-37.
1164. Barrie D, Hoffman PN, Wilson JA, Kramer JM. Contamination of hospital linen by *Bacillus cereus*. *Epidemiol Infect* 1994; 113: 297-306.
1165. Standaert SM, Hutcheson RH, Schaffner W. Nosocomial transmission of *Salmonella* gastroenteritis to laundry workers in a nursing home. *Infect Control Hosp Epidemiol* 1994; 15: 22-26.
1166. Pasternak J, Richtmann R, Ganme APP, et al. Scabies epidemic: Price and prejudice. *Infect Control Hosp Epidemiol* 1994; 15: 540-542.
1167. Loh W, Ng VV, Holton J. Bacterial flora on the white coats of medical students. *J Hosp Infect* 2000; 45: 65-68.
1168. Joint Committee on Healthcare Laundry Guidelines. *Guidelines for Healthcare Linen Service*. Textile Rental Service Association of America, Hallendale, FL, 1994.
1169. Greene VW. Microbiological contamination control in hospitals: Part 6 - roles of central service and the laundry. *Hospitals JAHA* 1970; 44: 98-103.
1170. Wagner RA. Partitioned laundry improves bacteria control. *Hospitals JAHA* 1966; 40: 148-151.
1171. Hambraeus A, Malmborg AS. Is a bed centre in a hospital a hygienic hazard? *J Hyg (Camb)* 1982; 88: 143-147.
1172. McDonald LL, Pugliese G. Textile processing service. In: *Hospital Epidemiology and Infection Control*, 2nd Ed., Mayhall CG, ed. Philadelphia PA; Lippincott Williams & Wilkins; 1999: p. 1031-1034.
1173. Legnani PP, Leoni E. Factors affecting the bacteriological contamination of commercial washing machines. *Zbl Hyg* 1997; 200: 319-333.
1174. Maki DG, Alvarado C, Hassemer C. Double-bagging of items from isolation rooms is unnecessary as an infection control measure: A comparative study of surface contamination with single- and double-bagging. *Infect Control* 1986; 7: 535-537.
1175. Garner JS, Simmons BP. CDC guideline for isolation precautions in hospitals. *Infect Control* 1983; 4: 245-325 and *Am J Infect Control* 1984; 12: 103-163.
1176. Weinstein SA, Gantz NM, Pelletier C, Hibert D. Bacterial surface contamination of patients' linen: Isolation precautions versus standard care. *Am J Infect Control* 1989; 17: 264-267.
1177. Hughes HG. Chutes in hospitals. *J Can Hosp Assoc* 1964; 41: 56-57.
1178. Michaelson GS. Designing linen chutes to reduce spread of infectious organisms. *Hospitals JAHA* 1965; 39: 116.
1179. Hoch KW. Laundry chute cleaning recommendations. *Infect Control* 1982; 3: 360.
1180. Whyte W, Baird G, Annand R. Bacterial contamination on the surface of hospital linen chutes. *J Hyg (Camb)*

1969; 67: 427-435.

1181. Taylor LJ. Segregation, collection, and disposal of hospital laundry and waste. *J Hosp Infect* 1988; 11 (Suppl. A): 57-63.

1182. Walter WG, Schillinger JE. Bacterial survival in laundered fabrics. *Appl Microbiol* 1975; 29: 368-373.

1183. Barrie D. How hospital linen and laundry services are provided. *J Hosp Infect* 1994; 27: 219-235.

1184. Mouton RP, Bekkers JH. Bacteriological results of routine procedures in a hospital laundry. *Folia Med Neerl* 1967; 10: 71-76.

1185. Nicholes PS. Bacteria in laundered fabrics. *Am J Public Health* 1970; 60: 2175-2180.

1186. Arnold L. A sanitary study of commercial laundry practices. *Am J Public Health* 1938; 28: 839-844.

1187. Belkin NL. Aseptics and aesthetics of chlorine bleach: Can its use in laundering be safely abandoned? *Am J Infect Control* 1998; 26: 149-151.

1188. Jordan WE, Jones DV. Antiviral effectiveness of chlorine bleach in household laundry use. *Am J Dis Child* 1969; 117: 313-316.

1189. Hittman Associates, Inc. Energy efficient water use in hospitals. Final summary report (H-W8000-78-756FR). 1979. Prepared for the University of California, Lawrence Berkeley Laboratory, Contract No. P.O. 4627702.

1190. Jaska JM, Fredell DL. Impact of detergent systems on bacterial survival on laundered fabrics. *Appl Environ Microbiol* 1980; 39: 743-748.

1191. Battles DR, Vesley D. Wash water temperature and sanitation in the hospital laundry. *J Environ Health* 1981; 43: 244-250.

1192. Christian RR, Manchester JT, Mellor MT. Bacteriological quality of fabrics washed a lower-than-standard temperatures in a hospital laundry facility. *Appl Environ Microbiol* 1983; 45: 591-597.

1193. Smith JA, Neil KR, Davidson CG, Davidson RW. Effect of water temperature on bacterial killing in laundry. *Infect Control* 1987; 8: 204-209.

1194. Tompkins DS, Johnson P, Fittall BR. Low-temperature washing of patients' clothing: Effects of detergent with disinfectant and a tunnel drier on bacterial survival. *J Hosp Infect* 1988; 12: 51-58.

1195. Ayliffe GAJ, Collins BJ, Taylor LJ. Laundering. In: *Hospital-Acquired Infection: Principles and Prevention*. Bristol, UK; Wright PSG; 1982: p. 101-106.

1196. Koller W, Wewalka G. A new method for microbiological evaluation of disinfecting laundering processes. *Zbl Bakt Hyg I Abt Orig B* 1982; 176: 463-471.

1197. Meyer CL, Eitzen HE, Schreiner RL, Gfell MA, Moye L, Kleiman MB. Should linen in newborn intensive care units be autoclaved? *Pediatrics* 1981; 67: 362-364.

1198. Wagg RE. Disinfection of textiles in laundering and dry cleaning. *Chem Ind* 1965; 44: 1830-1834.

1199. Bates CJ, Wilcox MH, Smith TL, Spencer RC. The efficacy of a hospital dry cleaning cycle in disinfecting material contaminated with bacteria and viruses. *J Hosp Infect* 1993; 23: 255-262.

1200. Oehnel E. Drycleaning in the hospital laundry. *Can Hosp* 1971; September: 66-68.

1201. DiGacomo JC, Odom JW, Ritoto PC, Swan KC. Cost containment in the operating room: Use of reusables versus disposable clothing. *Am Surg* 1992; 58: 654-656.

1202. American Society for Testing Materials. *Standard Test Method for Resistance of Materials Used in Protective Clothing to Penetration by Synthetic Blood*. American Society for Testing Materials. 1998; F1670-98.

1203. American Society for Testing Materials. *Standard Test Method for Resistance of Materials Used in Protective Clothing to Penetration by Bloodborne Pathogens Using Phi-X174 Bacteriophage Penetration as a Test System*. American Society for Testing Materials. 1997; F1671-976.

1204. Leonas KK, Jinkins RS. The relationship of selected fabric characteristics and the barrier effectiveness of surgical gown fabrics. *Am J Infect Control* 1997; 25: 16-23.

1205. Smith JW, Nichols RL. Barrier efficiency of surgical gowns. Are we really protected from our patients' pathogens? *Arch Surg* 1991; 126: 756-763.

1206. Leonas KK. Effect of laundering on the barrier properties of reusable surgical gown fabrics. *Am J Infect Control* 1998; 26: 495-501.

1207. McCullough EA. Methods for determining the barrier efficacy of surgical gowns. *Am J Infect Control* 1993; 21: 368-374.

1208. Pissiotis CA, Komborozos V, Papoutsi C, Skrekas G. Factors that influence the effectiveness of surgical gowns in the operating theater. *Eur J Surg* 1997; 163: 597-604.

1209. Association of Operating Room Nurses (AORN). Recommended practices for use and selection of barrier materials for surgical gowns and drapes. Association of Operating Room Nurses. *AORN J* 1996; 63: 650, 653-654.

1210. Murphy L. Cost/benefit study of reusable and disposable OR draping materials. *J Healthc Mater Manage* 1993; 11: 44-48.
1211. U.S. Environmental Protection Agency. Consumer Products Treated with Pesticides. Office of Pesticide Programs. <http://www.epa.gov/opp00001/citizens/treatart.htm>
1212. U.S. Environmental Protection Agency. Clarification of Treated Articles Exemption; Availability of Draft PR Notice. *Fed Reg* 1998; 63: 19256-19258.
1213. Mayer CE. FTC Challenges Antibacterial Product. *Washington Post*. September 17,1999: p. A09.
1214. Fujita K, Lilly HA, Kidson A, Ayliffe GAJ. Gentamicin-resistant *Pseudomonas aeruginosa* infection from mattresses in a burns unit. *Br Med J* 1981; 283: 219-220.
1215. Grubbs DJ, Watson KC. *Pseudomonas* septicemia from plastic mattresses. *Lancet* 1982; 1: 518.
1216. Sherertz RJ, Sullivan ML. An outbreak of infections with *Acinetobacter calcoaceticus* in burn patients: Contamination of patients' mattresses. *J Infect Dis* 1985; 151: 252-258.
1217. Ndawula EM, Brown L. Mattresses as reservoirs of epidemic methicillin-resistant *Staphylococcus aureus*. *Lancet* 1991; 337: 488.
1218. O'Donoghue MAT, Allen KD. Costs of an outbreak of wound infections in an orthopaedic ward. *J Hosp Infect* 1992; 22: 73-79.
1219. Weernink A, Severin WPJ, Thernberg T, Dijkshoorn L. Pillows, an unexpected source of *Acinetobacter*. *J Hosp Infect* 1995; 29: 189-199.
1220. Newsome TW, Johns LA, Pruitt BA Jr. Use of an air-fluidized bed in the care of patients with extensive burns. *Am J Surg* 1972; 124: 52-56.
1221. Scheidt A, Drusin LM. Bacteriologic contamination in an air-fluidized bed. *J Trauma* 1983; 23: 241-242.
1222. Freeman R, Gould FK, Ryan DW, Chamberlain J, Sisson PR. Nosocomial infection due to Enterococci attributed to a fluidized microsphere bed. The value of pyrolysis mass spectrometry. *J Hosp Infect* 1994; 27: 187-193.
1223. Sharbaugh RJ, Hargest TS. Bactericidal effect of the air-fluidized bed. *Am Surgeon* 1971; 37: 583-586.
1224. Sharbaugh RJ, Hargest TS, Wright FA. Further studies on the bactericidal effect of the air-fluidized bed. *Am Surgeon* 1973; 39: 253-256.
1225. Winters WD. A new perspective of microbial survival and dissemination in a prospectively contaminated air-fluidized bed model. *Am J Infect Control* 1990; 18: 307-315.
1226. Clancy MJ. Nosocomial infection and microsphere beds. *Lancet* 1993; 342: 680-681.
1227. Clancy MJ. Nosocomial infection due to Enterococci attributed to a fluidized microsphere bed. *J Hosp Infect* 1994; 28: 324-325.
1228. Vesley D, Hankinson SE, Lauer JL. Microbial survival and dissemination associated with an air-fluidized therapy unit. *Am J Infect Control* 1986; 14: 35-40.
1229. Bolyard EA, Townsend TR, Horan T. Airborne contamination associated with in-use air-fluidized beds: A descriptive study. *Am J Infect Control* 1987; 15: 75-78.
1230. Jacobsen E, Gurevich I, Cunha BA. Air-fluidized beds and negative-pressure isolation rooms. *Am J Infect Control* 1993; 21: 217-218.
1231. Cooper JE. Pets in hospitals. *Brit Med J*. 1976; 1: 698-700.
1232. Egerton JR. Pets and zoonoses. *Med J Aust* 1982; 2: 311.
1233. Yamauchi T. Pet programs in hospitals. *Pediatr Infect Dis J*. 1993; 12: 707.
1234. Khan MA, Farrag N. Animal-assisted activity and infection control implications in a healthcare setting. *J Hosp Infect* 2000; 46: 4-11.
1235. Weber DJ, Rutala WA. Epidemiology and prevention of nosocomial infections associated with animals in the hospital. In: *Hospital Epidemiology and Infection Control*, 2nd Ed, Mayhall CG, ed. Philadelphia PA; Lippincott Williams & Wilkins; 1999: p. 1399-1421.
1236. Acha PN, Szyfres B. *Zoonoses and Communicable Diseases Common to Man and Animals*, 2nd Ed, Scientific publication No. 503. Washington DC; Pan American Health Organization; 1987.
1237. Elliot DL, Tolle SW, Goldberg L, Miller JB. Pet-associated illness. *N Engl J Med* 1985; 313: 985-995.
1238. Marx MB. Parasites, pets, and people. *Primary Care* 1991; 18: 153-165.
1239. Goldstein EJ. Household pets and human infections. *Infect Dis Clin North Am* 1991; 5: 117-130.
1240. Chomel BB. Zoonoses of house pets other than dogs, cats, and birds. *Pediatr Infect Dis J* 1992; 11: 479-487.
1241. Gnann JW Jr, Bressler GS, Bodet CA III, Avent CK. Human blastomycosis after a dog bite. *Ann Intern Med* 1983; 98: 48-49.
1242. Garcia VF. Animal bites and *Pasteurella* infections. *Pediatr Rev*. 1997; 18: 127-130.

1243. Crowder HR, Dorn CR, Smith RE. Group A *Streptococcus* in pets and group A streptococcal disease in man. *Int J Zoonoses* 1978; 5: 45-54.
1244. Centers for Disease Control and Prevention. Reptile-associated salmonellosis - Selected states, 1996-1998. *MMWR* 1999; 48: 1009-1013.
1245. Devriese LA, Ieven M, Goossens H, et al. Presence of vancomycin-resistant enterococci in farm and pet animals. *Antimicrob Agent Chemother* 1996; 40: 2285-2287.
1246. Scott GM, Thomson R, Malone-Lee J, Ridgway GL. Cross-infection between animals and man: Possible feline transmission of *Staphylococcus aureus* infection in humans? *J Hosp Infect* 1988; 12: 29-34.
1247. Weinberg A. Ecology and epidemiology of zoonotic pathogens. *Infect Dis Clin North Am* 1991; 5: 1-6.
1248. Yu V, Meissner C. Zoonoses. In: *Mechanisms of Microbial Diseases*, Schaechter M, Medoff G, Schlessinger D, eds. Baltimore MD; Williams & Wilkins; 1989: p. 749-764.
1249. Ryan KJ. Some bacteria causing zoonotic diseases. In: *Medical Microbiology*, 2nd Ed., Sherris JC, ed. New York NY; Elsevier; 1990: p. 489-498.
1250. Chang HJ, Miller HL, Watkins N, et al. An epidemic of *Malassezia pachydermatis* in an intensive care nursery associated with colonization of healthcare workers' pet dog. *N Engl J Med* 1998; 338: 706-711.
1251. Richet HM, Craven PC, Brown JM, et al. A cluster of *Rhodococcus (Gordona) bronchialis* sternal-wound infections after coronary-artery bypass surgery. *N Engl J Med* 1991; 324: 104-109.
1252. Saylor K. Pet visitation program. *J Gerontol Nurs* 1998; 24: 36-38.
1253. Corson SA, O'Leary Corson E. Pets as mediators of therapy. *Curr Psychiatr Ther* 1978; 18: 195-205.
1254. Fick KM. The influence of an animal on social interactions of nursing home residents in a group setting. *Am J Occup Ther* 1993; 47: 529-534.
1255. Gunby P. Patient progressing well? He must have a pet. *JAMA* 1979; 241: 438.
1256. Culliton BJ. Take two pets and call me in the morning. *Science* 1987; 237: 1560-1561.
1257. Wilkes CN, Shalko TK, Trahan M. Pet Rx: Implications for good health. *Health Educ* 1989; 20: 6-9.
1258. Doyle K, Kukowski T. Utilization of pets in a hospice program. *Health Educ* 1989; 20: 10-11.
1259. Teeter LM. Pet therapy program. *JAVMA* 1997; 210: 1435-1438.
1260. Gammonley J, Yates J. Pet projects: Animal assisted therapy in nursing homes. *J Gerontol Nurs* 1991; 17: 12-15.
1261. Draper RJ, Gerber GJ, Layng EM. Defining the role of pet animals in psychotherapy. *Psychiat J Univ Ottawa* 1990; 15: 169-172.
1262. Allen DT. Effects of dogs on human health. *JAVMA* 1997; 210: 1136-1139.
1263. Delta Society. *Standards of Practice for Animal-Assisted Activities and Animal-Assisted Therapy*. Renton, WA: 1996.
1264. Centers for Disease Control and Prevention. *How To Prevent Transmission of Intestinal Roundworms from Pets to People*. <http://www.cdc.gov/ncidod/diseases/roundworm/roundworm.htm>
1265. American Academy of Allergy, Asthma, and Immunology. *Allergies to Animals* (brochure). Milwaukee WI; The Academy Press; 1995.
1266. Duncan SL, APIC Guideline Committee. APIC State-of-the-art report: The implications of service animals in healthcare settings. *Am J Infect Control* 2000; 28: 170-180.
1267. Murray AB, Ferguson A, Morrison BJ. The frequency and severity of cat allergy vs. dog allergy in atopic children. *J Allergy Clin Immunol* 1983; 72: 145-149.
1268. Hodson T, Custovic A, Simpson A, Chapman M, Woodcock A, Green R. Washing the dog reduces dog allergen levels, but the dog needs to be washed twice a week. *J Allergy Clin Immunol* 1999; 103: 581-585.
1269. Brickel CN. The therapeutic roles of cat mascots with a hospital based population: A staff survey. *Gerontologist* 1979; 19: 368-372.
1270. Thomas W, Stermer M. Eden alternative principles hold promise for the future of long-term care. *Balance* 1999; 3: 14-17.
1271. Tavormina CE. Embracing the Eden alternative in long-term care environments. *Geriatr Nurs* 1999; 20: 158-161.
1272. Brook I, Fish CH, Schantz PM, Cotton DD. Toxocariasis in an institution for the mentally retarded. *Infect Control* 1981; 2: 317-319.
1273. Huminer D, Symon R, Groskopf I, et al. Seroepidemiological study of toxocariasis and strongyloidiasis in adult mentally retarded institutionalized subjects. *Am J Trop Med Hyg* 1992; 46: 278-281.
1274. American with Disabilities Act; Public Law 101-336 (28 CFR 36.102 et seq.). Title III, Public Accommodations

Operated by Private Entities, Sect. 302, Prohibition of Discrimination by Public Accommodations; 42 USC 12182. July 26, 1990.

1275. Fox JG, Lipman NS. Infections transmitted by large and small laboratory animals. *Infect Dis Clin North Am* 1991; 5: 131-163.

1276. Department of Agriculture. Public Law 89-544 (The Animal Welfare Act of 1966). Title 7, United States Code, Sections 2131-2156.

1277. Department of Agriculture. Public Law 99-198 Food Security Act of 1985, Subtitle F - Animal Welfare. Title 7, United States Code, Section 2131.

1278. Althaus H, Sauerwald M, Schrammeck E. Waste from hospitals and sanatoria. *Zbl Bakteriol Hyg I Abt Orig B* 1983; 178: 1-29.

1279. Kalnowski G, Wiegand H, Henning R. The microbial contamination of hospital waste. *Zbl Bakteriol Hyg I Abt Orig B* 1983; 178: 364-379.

1280. Mose JR, Reinthaler F. Microbial contamination of hospital waste and household refuse. *Zbl Bakteriol Hyg I Abt Orig B* 1985; 181: 98-110

1281. Collins CH, Kennedy DA. The microbiological hazards of municipal and clinical wastes. *J Appl Bacteriol* 1992; 73: 1-6.

1282. Rutala WA, Odette RL, Samsa GP. Management of infectious waste by U.S. hospitals. *JAMA* 1989; 262: 1635-1640.

1283. Agency for Toxic Substances and Disease Registry. *The Public Health Implications of Medical Waste: A Report to Congress*. Department of Health and Human Services, Public Health Service, Atlanta, GA; 1990.

1284. Hedrick ER. Infectious waste management - Will science prevail? *Infect Control Hosp Epidemiol* 1988; 9: 488-490.

1285. Keene J. Medical waste management: Public pressure vs. sound science. *Hazard Mat Control* 1989; Sept/Oct: 29-36.

1286. Keene J. Medical waste: A minimal hazard. *Infect Control Hosp Epidemiol* 1991; 12: 682-685.

1287. Rutala WA, Weber DJ. Mismatch between science and policy. *N Engl J Med* 1991; 325: 578-582.

1288. U.S. Environmental Protection Agency. EPA Guide for Infectious Waste Management. EPA Publication No. 530SW86014; Washington DC; U.S. Government Printing Office: 1986.

1289. Greene R, Miele DJ, Slavik NS. *Technical Assistance Manual: State Regulatory Oversight of Medical Waste Treatment Technologies*, 2nd ed. A report of the State and Territorial Association on Alternative Treatment Technologies; 1994.

1290. U.S. Environmental Protection Agency (EPA). 40 CFR Part 60. Standards of Performance for New Stationary Sources and Emission Guidelines for Existing Sources: Hospital/Medical/Infectious Waste Incinerators; Final Rule. *Fed Reg* 1997; 62: 48347-48391.

1291. Centers for Disease Control/National Institutes of Health. *Biosafety in Microbiological and Biomedical Laboratories*. HHS Publication No. (CDC) 93-8395. Washington DC; U.S. Government Printing Office: 1985.

1292. Department of Health and Human Services, Centers for Disease Control and Prevention. Additional requirements for facilities transferring or receiving select agents; Final rule: 42 CFR Part 72. October 24, 1996. *Fed Reg* 1996; 61(207): 55189-55200.

1293. Centers for Disease Control. Recommendations on Infective Waste. Office of Biosafety and Hospital Infections Program; 1988: p. 1-6.

1294. Centers for Disease Control and Prevention. National Institute for Occupational Safety and Health. NIOSH Alert: Preventing Needlestick Injuries in Health Care Settings. DHHS-NIOSH Publication No. 2000-108; November 1999.

1295. Rutala WA, Stiegel MM, Sarubbi FA. Decontamination of laboratory microbiological waste by steam sterilization. *Appl Environ Microbiol* 1982; 43: 1311-1316.

1296. Lauer JL, Battles DR, Vesley D. Decontaminating infectious laboratory waste by autoclaving. *Appl Environ Microbiol* 1982; 44: 690-694.

1297. Palenik CJ, Cumberlander ND. Effects of steam sterilization on the contents of sharps containers. *Am J Infect Control* 1993; 21: 28-33.

1298. Weber AM, Boudreau Y, Mortimer VD. Stericycle, Inc., Morton, WA. HETA 98-0027-2709. NIOSH, CDC, Cincinnati, OH, 1998.

1299. Johnson KR, Braden CR, Cairns KL, et al. Transmission of *Mycobacterium tuberculosis* from medical waste. *JAMA* 2000; 284: 1683-1688.

1300. Emery R, Sprau D, Lao YJ, Pryor W. Release of bacterial aerosols during infectious waste compaction: An initial hazard evaluation for health care workers. *Am Ind Hyg Assoc J* 1992; 53: 339-345.
1301. National Committee for Clinical Laboratory Standards (NCCLS). 1997. Protection of laboratory workers from instrument biohazards and infectious disease transmitted by blood, body fluids, and tissue. Approved guideline. Dec. 1997, NCCLS Document M29-A (ISBN1-56238-339-6).
1302. Bond WW. Survival of hepatitis B virus in the environment. *JAMA* 1984; 252: 397-398.
1303. Slade JE, Pike EB, Eglin RP, Colbourne JS, Kurtz JB. The survival of human immunodeficiency virus in water, sewage, and sea water. *Water Sci Technol* 1989; 21: 55-59.
1304. Geertsma RE, Van Asten JAAM. Sterilization of prions. *Zentr Steril* 1995; 3: 385-394.
1305. Johnson MW, Mitch WE, Heller AH, Spector R. The impact of an educational program on gentamicin use in a teaching hospital. *Am J Med* 1982; 73: 9-14.
1306. Soumerai SB, Salem-Schatz S, Avorn J, Casteris CS, Ross-Degnan D, Popovsky MA. A controlled trial of educational outreach to improve blood transfusion practice. *JAMA* 1993; 270: 961-966.
1307. Eisenberg JM. An education program to modify laboratory use by house staff. *J Med Educ* 1977; 52: 578-581.
1308. Rello J, Quintana E, Ausina V, Puzo V, Puzo C, Net A, Prats G. Risk factors for *Staphylococcus aureus* nosocomial pneumonia in critically ill patients. *Am Rev Respir Dis* 1990; 142: 1320-1324.
1309. Aisner J, Murill J, Schimpff SC, Steere AC. Invasive aspergillosis in acute leukemia: Correlation with nose cultures and antibiotic use. *Ann Intern Med* 1979; 90: 4-9.
1310. Rogers TR. Infections in hematologic malignancy. *Infect Control* 1986; 7S: 124-125.
1311. McWhinney PHM, Kibbler CC, Hamon MD, et al. Progress in the diagnosis and management of aspergillosis in bone marrow transplantation: 13 years' experience. *J Infect Dis* 1993; 17: 397-404.
1312. Mutchler JE. Principles of ventilation. In: *NIOSH. The Industrial Environment - Its Evaluation and Control*. Washington DC; US Department of Health, Education, and Welfare, Public Health Service, NIOSH: 1973.
1313. Breiman RF, Cozen W, Fields BS, et al. Role of air sampling in investigation of an outbreak of Legionnaires' disease associated with exposure to aerosols from an evaporative condenser. *J Infect Dis* 1990; 161: 1257-1261.
1314. Centers for Disease Control and Prevention. Procedures for the recovery of *Legionella* from the environment. Atlanta GA; U.S. Department of Health and Human Services, Public Health Service, CDC; 1992: p 1-13.
1315. Costerton JW, Lewandowski Z, Caldwell DE, Korber DR, Lappin-Scott HM. Microbial biofilms. *Ann Rev Microbiol* 1995; 49: 711-745.
1316. LeChevallier MW, Babcock TM, Lee RG. Examination and characterization of distribution system biofilms. *Appl Environ Microbiol* 1987; 53: 2714-2724.
1317. Nagy LA, Olson BH. Occurrence and significance of bacteria, fungi, and yeasts associated with distribution pipe surfaces. *Proceeds of the Water Quality Technology Conference*; Portland OR; 1985: p. 213-238.
1318. Maki DG, Martin WT. Nationwide epidemic of septicemia caused by contaminated infusion products. IV growth of microbial pathogens in fluids for intravenous infusion. *J Infect Dis* 1975; 131: 267-272.
1319. Costerton JW, Stewart PS, Greenberg EP. Bacterial biofilms: A common cause of persistent infections. *Science* 1999; 284: 1318-1322.
1320. Costerton JW, Khoury AE, Ward KH, Anwar H. Practical measures to control device-related bacterial infections. *Int J Artif Organs* 1993; 16: 765-770.
1321. Nickel JC, Costerton JW, McLean RJC, Olson M. Bacterial biofilms: Influence on the pathogenesis, diagnosis, and treatment of urinary tract infections. *J Antimicrobial Chemother* 1994; 33 (Suppl. A): 31-41.
1322. LeChevallier MW, Cawthon CD, Lee RG. Inactivation of biofilm bacteria. *Appl Environ Microbiol* 1988; 54: 2492-2499.
1323. Anwar J, Strap JL, Costerton JW. Establishment of aging biofilms: Possible mechanism of bacterial resistance to antimicrobial therapy. *Antimicrobiol Agents Chemotherapy* 1992; 36: 1347-1351.
1324. Stewart PS. Biofilm accumulation model that predicts antibiotic resistance to *Pseudomonas aeruginosa* biofilms. *Antimicrobiol Agents Chemotherapy* 1994; 38: 1052-1058.
1325. Chen X, Stewart PS. Chlorine penetration into artificial biofilm is limited by a reaction-diffusion interaction. *Environ Sci Technol* 1996; 30: 2078-2083.
1326. Huang C-T, Yu FP, McFeters GA, Stewart PS. Nonuniform spatial patterns of respiratory activity with biofilms during disinfection. *Appl Environ Microbiol* 1995; 61: 2252-2256.
1327. Donlan RM, Pipes WO. Selected drinking water characteristics and attached microbial population density. *J AWWA* 1988; 80: 70-76.
1328. Reasoner DJ, Geldreich EE. A new medium for the enumeration and subculture of bacteria from potable water.

Appl Environ Microbiol 1985; 49: 1-7.

1329. Pass T, Wright R, Sharp B, Harding GB. Culture of dialysis fluids on nutrient-rich media for short periods at elevated temperatures underestimates microbial contamination. *Blood Purif* 1996; 14: 136-145.

1330. Arduino MJ, Bland LA, Agüero SM, et al. Effects of incubation time and temperature on microbiologic sampling procedures for hemodialysis fluids. *J Clin Microbiol* 1991; 29: 1462-1465.

1331. Pearson FC, Weary ME, Sargent HE, et al. Comparison of several control standard endotoxins to the National Reference Standard Endotoxin - An HIMA collaborative study. *Appl Environ Microbiol* 1985; 50: 91-93.

1332. Arnow PM, Weil D, Para MF. Prevalence of significance of *Legionella pneumophila* contamination of residential hot-tap water systems. *J Infect Dis* 1985; 152: 145-151.

1333. Shelton BG, Morris GK, Gorman GW. Reducing risks associated with *Legionella* bacteria in building water systems. In: *Legionella: Current Status and Emerging Perspectives*, Barbaree JM, Breiman RF, Dufour AP, eds. Washington DC; American Society for Microbiology Press; 1993: p. 279-281.

1334. Joly JR. Monitoring for the presence of *Legionella*: Where, when, and how? In: *Legionella: Current Status and Emerging Perspectives*, Barbaree JM, Breiman RF, Dufour AP, eds. Washington DC; American Society for Microbiology Press; 1993: p. 211-216.

1335. Brenner DJ, Feeley JC, Weaver RE. Family VII. *Legionellaceae*. In: *Bergey's Manual of Systemic Bacteriology, Vol 1*, Krieg NR, Holt JG, eds. Baltimore MD; Williams & Wilkins; 1984: p. 279.

1336. Katz SM, Hammel JM. The effect of drying, heat, and pH on the survival of *Legionella pneumophila*. *Ann Clin Lab Sci* 1987; 17: 150-156.

1337. Alary MA, Joly JR. Comparison of culture methods and an immunofluorescence assay for the detection of *Legionella pneumophila* in domestic hot water devices. *Curr Microbiol* 1992; 25: 19-25.

1338. Vickers RM, Stout JE, Yu VL. Failure of a diagnostic monoclonal immunofluorescent reagent to detect *Legionella pneumophila* in environmental samples. *Appl Environ Microbiol* 1990; 56: 2912-2914.

1339. Flournoy DJ, Belobraydic KA, Silberg SL, Lawrence CH, Guthrie PJ. False positive *Legionella pneumophila* direct immunofluorescence monoclonal antibody test caused by *Bacillus cereus* spores. *Diag Microbiol Infect Dis* 1988; 9: 123-125.

1340. Bej AK, Majbubani MH, Atlas RM. Detection of viable *Legionella pneumophila* in water by polymerase chain reaction and gene probe methods. *Appl Environ Microbiol* 1991; 57: 597-600.

1341. Schulze-Röbbecke R, Jung KD, Pullman H, Hundgeburth J. Control of *Legionella pneumophila* in a hospital hot water system. *Zbl Hyg* 1990; 190: 84-100.

1342. Colbourne JS, Pratt DJ, Smith MG, Fisher-Hoch SP, Harper D. Water fittings as sources of *Legionella pneumophila* in a hospital plumbing system. *Lancet* 1984; 1: 210-213.

1343. U.S. Environmental Protection Agency. National interim primary drinking water regulations: Control of trihalomethanes in drinking water: Final rules. *Federal Register* 1979; 44: 68624-68705.

1344. U.S. Environmental Protection Agency. National interim primary drinking water regulations: Trihalomethanes. *Federal Register* 1983; 48: 8406-8414.

IV Appendices

Appendix A - Glossary of Terms

This glossary contains many of the terms used in this guideline, as well as others that are encountered frequently when implementing these control measures. The definitions are generally not dictionary definitions, but are those most applicable to environmental infection control situations.

Acceptable indoor air quality - air in which there are no known contaminants at harmful concentrations as determined by knowledgeable authorities and with which a substantial majority ($\geq 80\%$) of the people exposed do not express dissatisfaction.

ACGIH - American Conference of Governmental Industrial Hygienists.

Aerosol - particles of respirable size generated by both humans and environmental sources and that have the capability of remaining viable and airborne for extended periods in the indoor environment.

AIA - American Institute of Architects: professional group responsible for publishing the "Guidelines for Design and Construction of Hospitals and Healthcare Facilities," a consensus document for design and construction of health care facilities endorsed by the U.S. Department of Health and Human Services, healthcare professionals, and professional organizations.

Air changes per hour (ACH) - the ratio of the volume of air flowing through a space in a certain period of time (i.e., the airflow rate) to the volume of that space (i.e., the room volume); this ratio is usually expressed as the number of air changes per hour (ACH).

Air mixing - the degree to which air supplied to a room mixes with the air already in the room, usually expressed as a mixing factor. This factor varies from 1 (for perfect mixing) to 10 (for poor mixing), and it is used as a multiplier to determine the actual airflow required (i.e., the recommended ACH multiplied by the mixing factor equals the actual ACH required).

Airborne transmission - a means of spreading infection when airborne droplet nuclei (small particle residue of evaporated droplets $\leq 5 \mu\text{m}$ in size containing microorganisms that remain suspended in air for long periods of time) are inhaled by the susceptible host.

Air-cleaning system - a device or combination of devices applied to reduce the concentration of airborne contaminants (i.e., microorganisms, dusts, fumes, aerosols, other particulate matter, gases).

Air conditioning - the process of treating air to meet the requirements of a conditioned space by controlling its temperature, humidity, cleanliness, and distribution.

Allogeneic - non-twin, non-self; refers to transplanted tissue from a donor closely matched to a recipient but not related to that person.

Ambient air - the air surrounding an object.

Anemometer - a flow meter which measures the wind force and velocity of air. An anemometer is often used as a means of determining the volume of air being drawn into an air sampler.

Anteroom - a small room leading from a corridor into an isolation room: this room can act as an airlock, preventing the escape of contaminants from the isolation room into the corridor.

ASHAE - American Society of Hospital Engineers, an association affiliated with the American Hospital Association.

ASHRAE - American Society of Heating, Refrigerating, and Air Conditioning Engineers Inc. The engineering counterpart of AIA.

Autologous - self; refers to transplanted tissue whose source is the same as the recipient, or a twin.

Automated cyler - a machine used during peritoneal dialysis which pumps fluid into and out of the patient while he/she sleeps.

Biochemical oxygen demand (BOD) - a measure of the amount of oxygen removed from aquatic environments by aerobic microorganisms for their metabolic requirements. Measurement of BOD is used to determine the level of organic pollution of a stream or lake. The greater the BOD, the greater the degree of water pollution. Also referred to as Biological Oxygen Demand (BOD).

Biological oxygen demand (BOD) - as this pertains to water quality, an indirect measure of the concentration of biologically degradable material present in organic wastes. It usually reflects the amount of oxygen consumed in five days by biological processes breaking down organic waste (BOD5).

Biosafety level - a combination of microbiological practices, laboratory facilities, and safety equipment determined to be sufficient to reduce or prevent occupational exposures of laboratory personnel to the microbiological agents they work

with. There are four biosafety levels based on the hazards associated with the various microbiological agents.

BOD5 - the amount of dissolved oxygen consumed in five days by biological processes breaking down organic matter.

Bonneting - a floor cleaning method for either carpeted or hard surface floors which uses a circular motion of a large, fibrous disc to lift soil and dust from the surface and remove it.

Capped spur - a pipe leading from the water recirculating system to an outlet that has been closed off ("capped"). A capped spur cannot be flushed, and it might not be noticed unless the surrounding wall is removed.

CFU/m³ - colony forming units per cubic meter (of air)

Chlamydo spores - thick-walled, typically spherical or ovoid resting spores produced (asexually) by certain types of fungi from cells of the somatic hyphae.

Chloramines - compounds containing nitrogen, hydrogen, and chlorine, formed by the reaction between hypochlorous acid (HOCl) and ammonia (NH₃) and/or organic amines in water. The formation of chloramines in drinking water treatment extends the disinfecting power of chlorine. Also referred to as Combined Available Chlorine.

Cleaning - the removal of visible soil and organic contamination from a device or surface, using either the physical action of scrubbing with a surfactant or detergent and water, or an energy-based process (e.g., ultrasonic cleaners) with appropriate chemical agents.

Coagulation-flocculation - coagulation is the clumping of particles which results in the settling of impurities. It may be induced by coagulants such as lime, alum, and iron salts. Flocculation in water and wastewater treatment is the agglomeration or clustering of colloidal and finely divided suspended matter after coagulation by gentle stirring by either mechanical or hydraulic means such that they can be separated from water or sewage.

Commissioning (a room) - testing a system or device to ensure that it meets the pre-use specifications as indicated by the manufacturer or predetermined standard, or air sampling in a room to establish a pre-occupancy baseline standard of microbial or particulate contamination. Also referred to as benchmarking at 25°C.

Conidia - asexual spores of fungi borne externally.

Conidiophores - specialized hyphae that bear conidia in fungi.

Conditioned space - that part of a building that is heated or cooled, or both, for the comfort of the occupants.

Contaminant - an unwanted airborne constituent that may reduce acceptability of the air.

Convection - the transfer of heat or other atmospheric properties within the atmosphere or in the airspace of an enclosure by the circulation of currents from one region to another, especially by such motion directed upward.

Cooling tower - a structure engineered to receive accumulated heat from ventilation systems and equipment and transfer this heat to water, which then releases the stored heat to the atmosphere through evaporative cooling.

Critical item (medical instrument) - medical instruments or devices that contact normally sterile areas of the body or enter the vascular system. There is a high risk of infection from these devices if these are microbiologically contaminated prior to use; these devices must be sterile before use.

Dead legs - areas in the water system where water stagnates. A dead leg is a pipe, or spur, leading from the water recirculating system to an outlet that is used infrequently, resulting in inadequate flow of heat or chlorine from the recirculating system to the outlet.

Deionization - removal of ions from water by exchange with other ions associated with fixed charges on a resin bed. Cations are usually removed and H⁺ ions are exchanged; OH⁻ ions are exchanged for anions.

Detritus - particulate matter produced by or remaining after the wearing away or disintegration of a substance or tissue.

Dew point - the temperature at which a gas or vapor condenses to form a liquid; the point at which moisture begins to condense out of the air. At dew point, air is cooled to the point where it is at 100% relative humidity or saturation.

Dialysate - the aqueous electrolyte solution, usually containing dextrose, used to make a concentration gradient between the solution and blood in the hemodialyzer (dialyzer).

Dialyzer - a device that consists of two compartments (blood and dialysate) separated by a semipermeable membrane. A dialyzer is usually referred to as an artificial kidney.

Diffuser - the grille plate which disperses the air stream coming into the conditioned air space.

Direct transmission - involves direct body surface-to-body surface contact and physical transfer of microorganisms between a susceptible host and an infected/colonized person, or exposure to cloud of infectious particles within 3 feet; particles are >5 μm in size.

Disability - as defined by the Americans with Disabilities Act, is any physical or mental impairment that substantially limits one or more major life activities, including but not limited to walking, talking, seeing, breathing, hearing, or caring for oneself.

Disinfection - a generally less lethal process of microbial inactivation (compared to sterilization) which eliminates virtually all recognized pathogenic microorganisms but not necessarily all microbial forms (e.g., bacterial spores).

Drain pans - collect water as air and steam result in condensation.

Drift - circulating water lost from the cooling tower as liquid droplets entrained in the exhaust air stream (i.e., exhaust aerosols from a cooling tower).

Drift eliminators - an assembly of baffles or labyrinth passages through which the air passes prior to its exit from the cooling tower, for the purpose of removing entrained water droplets from the exhaust air.

Droplets - particles of moisture, such as are generated when a person coughs or sneezes, or when water is converted to a fine mist by a device such as an aerator or shower head. Intermediate in size between drops and droplet nuclei, these particles, although they may still contain infectious microorganisms, tend to quickly settle out from the air so that any risk of disease transmission is generally limited to persons in close proximity to the droplet source.

Droplet nuclei - sufficiently small particles (1 - 5 μ m in diameter) that can remain airborne indefinitely and cause infection when a susceptible person is exposed at or beyond 3 feet of particle source.

Dual duct system - an HVAC system that consists of parallel ducts that produce a cold air stream in one and a hot air stream in the other.

Dust - an air suspension of particles (aerosol) of any solid material, usually with particle sizes \leq 100 μ m in diameter.

Dust spot test - a procedure which uses atmospheric air or a defined dust to measure a filter's ability to remove particles. A photometer is used to measure air samples on either side of the filter, and the difference is expressed as a percentage of particles removed.

Effective leakage area - the area through which air can enter or leave the room. This does not include supply, return, or exhaust ducts. The smaller the effective leakage area, the better isolated the room.

Endotoxin - the lipopolysaccharides of gram-negative bacteria, the toxic character of which resides in the lipid portion. Endotoxins generally produce pyrogenic reactions in persons exposed to these bacterial components.

Enveloped virus - a virus whose outer surface is derived from a membrane of the host cell (either nuclear or outer membrane) during the budding phase of the maturation process. This membrane-derived material contains lipids, which makes these viruses sensitive to the action of chemical germicides.

Evaporative condenser - a wet-type, heat-rejection unit that produces large volumes of aerosols during the process of removing heat from conditioned space air.

Exhaust air - air removed from a space and not reused therein.

Exposure - the condition of being subjected to something (e.g., infectious agents) that could have a harmful effect.

Fastidious - having complex nutritional requirements for growth, as in microorganisms.

Fill - that portion of a cooling tower which makes up its primary heat transfer surface. Fill is alternatively known as "packing."

Finished water - treated, or potable water.

Fixed room-air HEPA recirculation systems - nonmobile devices or systems that remove airborne contaminants by recirculating air through a HEPA filter. These may be built into the room and permanently ducted or may be mounted to the wall or ceiling within the room. In either situation, they are fixed in place and are not easily movable.

Fomites - an inanimate object that may be contaminated with microorganisms and serve in their transmission.

Free, available chlorine - the term applied to the three forms of chlorine that may be found in solution (Cl_2 , OCl^- , and HOCl).

Germicide - a chemical that destroys microorganisms. Germicides may be used to inactivate microorganisms in or on living tissue (antiseptics) or on environmental surfaces (disinfectants).

Healthcare-associated - an outcome, usually an infection, that occurs in any healthcare facility as a result of medical care. The term "healthcare-associated" replaces "nosocomial," the latter term being limited to adverse infectious outcomes occurring in hospitals only.

Hemodiafiltration - a form of renal replacement therapy in which waste solutes in the patient's blood are removed by both diffusion and convection through a high-flux membrane.

Hemodialysis - a treatment for renal replacement therapy in which waste solutes in the patient's blood are removed by diffusion and/or convection through the semi-permeable membrane of an artificial kidney or dialyzer.

Hemofiltration - cleansing of waste products or other toxins from the blood by convection across a semi-permeable high-flux membrane where fluid balance is maintained by infusion of sterile, pyrogen-free substitution fluid pre- or post-hemodialyzer.

HEPA filter - High Efficiency Particulate Air filters capable of removing 99.97% of particles

$\geq 0.3 \mu\text{m}$ in diameter and may assist in controlling the transmission of airborne disease agents. These filters may be used in ventilation systems to remove particles from the air or in personal respirators to filter air before it is inhaled by the person wearing the respirator. The use of HEPA filters in ventilation systems requires expertise in installation and maintenance. To test this type of filter, $0.3 \mu\text{m}$ particles of dioctylphthalate (DOP) are drawn through the filter. Efficiency is calculated by comparing the downstream and upstream particle counts. The optimal HEPA filter allows only three particles to pass through for every 10,000 particles that are fed to the filter.

Heterotrophic (heterotroph) - that which requires some nutrient components from exogenous sources. Heterotrophic bacteria cannot synthesize all of their metabolites and therefore require certain nutrients from other sources.

High efficiency filter - a filter with a particle-removal efficiency of 90% - 95%.

High flux - type of dialyzer or hemodialysis treatment in which large molecules ($>8,000$ daltons [e.g., β_2 microglobulin]) are removed.

High-level disinfection - a disinfection process which inactivates vegetative bacteria, mycobacteria, fungi, and viruses, but not necessarily high numbers of bacterial spores.

Housekeeping surfaces - environmental surfaces (e.g., floors, walls, ceilings, tabletops) which are not involved in direct delivery of patient care in healthcare facilities

Hoyer lift - an apparatus which facilitates the repositioning of the non-ambulatory patient from bed to wheelchair or gurney and subsequently to therapy equipment (i.e., immersion tanks).

Hubbard tank - a tank used in hydrotherapy which may accommodate whole-body immersion, such as may be indicated for burn therapy. Use of a Hubbard tank has largely been replaced by bedside post-lavage therapy for wound care management.

HVAC - Heating, Ventilation, Air Conditioning.

Iatrogenic - induced in a patient by a physician's activity, manner, or therapy. Used especially in reference to an infectious disease or other complication of medical treatment.

Impactor - an air sampling device in which particles and microorganisms are directed onto a solid surface and retained there for assay.

Impingement - an air sampling method during which particles and microorganisms are directed into a liquid and retained there for assay.

Indirect transmission - involves contact of a susceptible host with a contaminated intermediate object, usually inanimate.

Induction unit - the terminal unit of an in-room ventilation system. Induction units take centrally conditioned air and further moderate its temperature. Induction units are not appropriate for areas with high exhaust requirements (e.g., research laboratories).

Intermediate-level disinfection - a disinfection process which inactivates vegetative bacteria, most fungi, mycobacteria, and most viruses (particularly the enveloped viruses), but does not inactivate bacterial spores.

Isoform - a possible configuration of a protein molecule, with a particular tertiary structure. With CJD prion proteins, for example, the molecules with large amounts of β -conformation are the normal isoform or version of that particular protein, whereas those prions with large amounts of β -sheet conformation are the proteins associated with the development of spongiform encephalopathy.

Laminar flow - HEPA filtered air that is blown into a room at a rate of 90 ± 10 feet/min in a unidirectional pattern with 100 - 400 ACH.

Large enveloped virus - viruses whose particle diameter is greater than 50 nm and whose outer surface is covered by a lipid-containing structure derived from the membranes of the host cells. Examples of large enveloped viruses include influenza viruses, herpes simplex viruses, poxviruses. **Laser plume** - the transfer of electromagnetic energy into tissues which results in a release of particles, gases, and tissue debris.

Lipid-containing viruses - viruses whose particle contains lipid components. The term is roughly synonymous with enveloped viruses whose outer surface is derived from host cell membranes. Lipid-containing viruses are sensitive to the inactivating effects of liquid chemical germicides.

Lithotriptors - instruments used for crushing calculi (i.e., stones, sand) in the bladder or kidneys.

Low efficiency filter - the prefilter with a particle-removal efficiency of approximately 30% through which incoming air first passes. See also Prefilter.

Low-level disinfection - a disinfection process which will inactivate most vegetative bacteria, some fungi, and some viruses, but cannot be relied on to inactivate resistant microorganisms (e.g., mycobacteria or bacterial spores).

Makeup air - outdoor air supplied to replace exhaust air and filtration.

Makeup water - cold water supply source for a cooling tower.

Manometer - a device which measures the pressure of liquids and gases. A manometer is commonly used to verify air filter performance by measuring pressure differentials on either side of the filter.

Membrane filtration - an assay method suitable for recovery and enumeration of microorganisms from liquid samples.

Mesophilic - that which favors a moderate temperature. For mesophilic bacteria, a temperature range of 20°C - 55°C (68°F - 131°F) is favorable for their growth and proliferation.

Mixing box - site where the cold and hot air streams mix in the HVAC system, usually situated close to the air outlet for the room.

Mixing faucet - a faucet which mixes hot and cold water to produce water at a desired temperature.

MMAD - Mass Median Aerodynamic Diameter: the unit used by ACGIH to describe the size of particles when particulate air sampling is conducted.

Moniliaceous - hyaline or brightly colored; laboratory terminology for the distinctive characteristics of certain opportunistic fungi in culture (e.g., *Aspergillus* spp., *Fusarium* spp.).

Monochloramine - the result of the reaction between chlorine and ammonia that contains only one chlorine atom.

Natural ventilation - the movement of outdoor air into a space through intentionally provided openings (i.e., windows, doors, nonpowered ventilators).

Negative pressure - air pressure differential between two adjacent airspaces such that airflow is directed into the room relative to the corridor ventilation (i.e., room air is prevented from flowing out of the room and into adjacent areas).

Neutropenia - a medical condition in which the patient's concentration of neutrophils is substantially less than that in the normal range. Severe neutropenia occurs when the concentration is <1,000 polymorphonuclear cells/μL for 2 weeks or <100 polymorphonuclear cells /mL for 1 week, particularly for hematopoietic stem cell transplant (HSCT) recipients.

Non-critical devices - these medical devices or surfaces come into contact with only intact skin. The risk of infection from using these devices is low.

Non-enveloped virus - a virus whose particle is not covered by a structure derived from a membrane of the host cell. Non-enveloped viruses have little or no lipid compounds in their biochemical composition, which is significant to their inherent resistance to the action of chemical germicides.

Nosocomial - an occurrence, usually an infection, that is acquired in a hospital as a result of medical care.

Nuisance dust - generally innocuous dust, not recognized as the direct cause of serious pathological conditions.

Oocysts - a cyst in which sporozoites are formed; a reproductive aspect of the life cycle of a number of parasitic agents (i.e., *Cryptosporidium* spp., *Cyclospora* spp.)

Outdoor air - air taken from the external atmosphere and, therefore, not previously circulated through the system.

Parallel streamlines - a unidirectional airflow pattern achieved in a laminar flow setting, characterized by little or no mixing of air.

Particulate matter (particles) - a state of matter in which solid or liquid substances exist in the form of aggregated molecules or particles. Airborne particulate matter is typically in the size range of 0.01 - 100 μm diameter.

Pasteurization - a disinfecting method for liquids during which the liquids are heated to 60°C (140°F) for a short time (≥30 mins.) to significantly reduce the numbers of pathogenic or spoilage microorganisms.

Plinth - a treatment table, a piece of equipment used to reposition the patient for treatment.

Portable room-air HEPA recirculation units - free-standing portable devices that remove airborne contaminants by recirculating air through a HEPA filter.

Positive pressure - air pressure differential between two adjacent air spaces such that airflow is directed from the room relative to the corridor ventilation (i.e., air from corridors, adjacent areas is prevented from entering the room).

Potable (drinking) water - water that is fit to drink. The microbiological quality of this water as defined by EPA microbiological standards from the Surface Water Treatment Rule: 1) *Giardia lamblia*: 99.9% killed/inactivated; 2) viruses: 99.9% inactivated; 3) *Legionella* spp.: no limit, but if *Giardia* and viruses are inactivated, *Legionella* will also be controlled; 4) heterotrophic plate count [HPC]: ≤ 500 CFU/mL; and 5) > 5% of water samples total coliform-positive in a month.

PPE - Personal Protective Equipment

ppm - parts per million. A measure of concentration in solution. A 5.25% chlorine bleach solution (undiluted as supplied by the manufacturer) contains approximately 50,000 parts per million of free available chlorine.

Prefilter - the first filter for incoming fresh air in a HVAC system that is approximately 30% efficient in removing particles from the air. See also low-efficiency filter.

Prion - a class of agents associated with the transmission of diseases known as transmissible spongiform encephalopathies (TSEs). Prions are considered to consist of protein only, and the abnormal isoform of this protein is thought to be the agent which causes diseases such as Creutzfeldt-Jakob disease (CJD), kuru, scrapie, bovine spongiform

encephalopathy (BSE), and the human version of BSE which is variant CJD (vCJD).

Pseudoepidemic (pseudo-outbreak) - a cluster of positive microbiologic cultures in the absence of clinical disease that results from contamination of the laboratory apparatus and process used to recover microorganisms.

Pyrogenic - an endotoxin burden such that a patient would receive ≥ 5 endotoxin units (EU) per kilogram of body weight per hour, thereby causing a febrile response. In dialysis this usually refers to water or dialysate having endotoxin concentrations of ≥ 5 EU/mL.

Rank order - a strategy for assessing overall indoor air quality and filter performance by comparing airborne particle counts from highest to lowest (i.e., from the best filtered air spaces to those with the least filtration).

RAPD - genotyping microorganisms by randomly amplified polymorphic DNA, a method of polymerase chain reaction.

Recirculated air - air removed from the conditioned space and intended for reuse as supply air.

Relative humidity - the ratio of the amount of water vapor in the atmosphere to the amount necessary for saturation at the same temperature. Relative humidity is expressed in terms of percent and measures the percentage of saturation. At 100% relative humidity, the air is saturated. The relative humidity decreases when the temperature is increased without changing the amount of moisture in the air.

Reprocessing (of medical instruments) - the procedures or steps taken to make a medical instrument safe for use on the next patient. Reprocessing encompasses both cleaning and the final or terminal step (i.e., sterilization or disinfection) which is determined by the intended use of the instrument according to the Spaulding classification.

Residuals - the presence and concentration of a chemical in media (e.g., water) or on a surface after the chemical has been added.

Reservoir - a nonclinical source of infection.

Respirable particles - those particles that penetrate into and are deposited in the nonciliated portion of the lung. Particles $> 10 \mu\text{m}$ in diameter are not respirable.

Return air - air removed from a space to be then recirculated or exhausted.

Reverse-osmosis (RO) - an advanced method of water or wastewater treatment that relies on a semi-permeable membrane to separate waters from pollutants. An external force is used to reverse the normal osmotic process resulting in the solvent moving from a solution of higher concentration to one of lower concentration.

Riser - water piping which connects the circulating water supply line, from the level of the base of the tower or supply header, to the tower's distribution system.

RODAC - Replicate Organism Direct Agar Contact. A nutrient agar plate whose convex agar surface is directly pressed onto an environmental surface for the purpose of microbiologic sampling of that surface.

Room-air HEPA recirculation systems and units - devices (either fixed or portable) that remove airborne contaminants by recirculating air through a HEPA filter.

Routine sampling - environmental sampling conducted without a specific, intended purpose and with no action plan dependent on the results obtained.

Sanitizer - an agent that reduces microbial contamination to safe levels as judged by public health standards or requirements.

Saprophytic - a naturally-occurring microbial contaminant.

Sedimentation - the act or process of depositing sediment from suspension in water, letting solids settle out of wastewater by gravity during treatment.

Semi-critical devices - medical devices that come into contact with mucous membranes or non-intact skin.

Service animal - any animal individually trained to do work or perform tasks for the benefit of a person with a disability.

Shedding - generation of particles and spores by sources within the patient area, such as patient movement and airflow over surfaces.

Single-pass ventilation - ventilation in which 100% of the air supplied to an area is exhausted to the outside.

Small, non-enveloped viruses - viruses whose particle diameter is less than 50 nm and whose outer surface is the protein of the particle itself and not that of host cell membrane components. Examples of small, non-enveloped viruses are polioviruses, hepatitis A virus.

Spaulding Classification - the categorization of inanimate surfaces in the medical environment as proposed in 1972 by Dr. Earle Spaulding. Surfaces are divided into three general categories, based on the theoretical risk of infection if the surfaces are contaminated at time of use. The categories are "critical," "semi-critical," and "non-critical."

Specific humidity - the mass of water vapor per unit mass of moist air. It is usually expressed as grains of water per pound of dry air, or pounds of water per pound of dry air. The specific humidity changes as moisture is added or removed. However, temperature changes do not change the specific humidity unless the air is cooled below the dew

point.

Splatter - visible drops of liquid or body fluid which are expelled forcibly into the air and settle out quickly, as distinguished from particles of an aerosol which remain airborne indefinitely.

Steady state - the usual state of an area.

Sterilization - the use of a physical or chemical procedure to destroy all microbial life, including large numbers of highly resistant bacterial endospores.

Stop valve - a valve that regulates the flow of fluid through a pipe; a faucet.

Substitution fluid - fluid which is used for fluid management of patients receiving hemodiafiltration. This fluid can be prepared on-line at the machine through a series of ultrafilters or with the use of sterile peritoneal dialysis fluid.

Supply air - that air delivered to the conditioned space and used for ventilation, heating, cooling, humidification, or dehumidification.

Tensile strength - the resistance of a material to a force tending to tear it apart, measured as the maximum tension the material can withstand without tearing.

Therapy animal - an animal, usually a personal pet that, with their owners, provide supervised, goal-directed intervention to clients in hospitals, nursing homes, special-population schools, and other treatment sites.

Thermophilic - capable of growing in environments warmer than body temperature.

Thermotolerant - capable of withstanding high temperature conditions.

TLV® - An exposure level under which most people can work consistently for 8 hours a day, day after day, without adverse effects. Used by the ACGIH to designate degree of exposure to contaminants. TLV® can be expressed as approximate milligrams of particulate per cubic meter of air (mg/m^3). TLVs® are listed as either an 8-hour TWA (time weighted average) or a 15-minute STEL (short term exposure limit).

TLV-TWA - Threshold Limit Value-Time Weighted Average: the time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be exposed repeatedly, day after day, without adverse effects. The TLV-TWA for “particulates (insoluble) not otherwise classified” (PNOC) - (sometimes referred to as nuisance dust) - are those particulates containing no asbestos and <1% crystalline silica. A TLV-TWA of 10 mg/m^3 for inhalable particulates and a TLV-TWA of 3 mg/m^3 for respirable particulates (particulates $\leq 5 \mu\text{m}$ in aerodynamic diameter) have been established.

Total suspended particulate matter - the mass of particles suspended in a unit of volume of air when collected by a high-volume air sampler.

Transient - a change in the condition of the steady state that takes a very short time compared with the steady state. Opening a door, and shaking bed linens are examples of transients.

TWA - Average exposure for an individual over a given working period, as determined by sampling at given times during the period. TWA is usually presented as the average concentration over an 8-hour workday for a 40-hour workweek.

Ultraclean air - air in laminar flow ventilation which has also passed through a bank of HEPA filters.

Ultrafiltered dialysate - the process by which dialysate is passed through a filter having a molecular weight cut-off of approximately 1 kilodalton for the purpose of removing bacteria and endotoxin from the bath.

Ultraviolet germicidal irradiation (UVGI) - the use of ultraviolet radiation to kill or inactivate microorganisms.

Ultraviolet germicidal irradiation lamps - lamps that kill or inactivate microorganisms by emitting ultraviolet germicidal radiation, predominantly at a wavelength of 254 nm. UVGI lamps can be used in ceiling or wall fixtures or within air ducts of ventilation systems.

Vapor pressure - the pressure exerted by free molecules at the surface of a solid or liquid. Vapor pressure is a function of temperature - it increases as the temperature increases.

Vegetative bacteria - bacteria which are actively growing and metabolizing, as opposed to a bacterial state of quiescence which is achieved when certain bacteria (i.e., gram-positive bacilli) convert to spores when the environment can no longer support active growth.

Vehicle - any object, person, surface, fomite, or media which may carry and transfer infectious microorganisms from one site to another.

Ventilation - the process of supplying and removing air by natural or mechanical means to and from any space. Such air may or may not be conditioned.

Ventilation air - that portion of the supply air that is outdoor air plus any recirculated air that has been treated for the purpose of maintaining acceptable indoor air quality.

Ventilation, dilution - an engineering control technique to dilute and remove airborne contaminants by the flow of air into and out of an area. Air that contains droplet nuclei is removed and replaced by contaminant-free air. If the flow is

sufficient, droplet nuclei become dispersed, and their concentration in the air is diminished.

Ventilation, local exhaust - ventilation used to capture and removed airborne contaminants by enclosing the contaminant source (i.e., the patient) or by placing an exhaust hood close to the contaminant source.

v/v - volume to volume. An expression of concentration of a percentage solution when the principle component is added as a liquid to the diluent.

w/v - weight to volume. An expression of concentration of a percentage solution when the principle component is added as a solid to the diluent.

Weight-arrestance - a measure of filter efficiency, used primarily when describing the performance of low- and medium-efficiency filters. A standardized synthetic dust is fed to the filter, and the weight fraction of the dust removed is determined.

Appendix B - Air

1. Removal of Airborne Contaminants

Table B.1. Air Changes per Hour (ACH) and Time in Minutes Required for Removal Efficiencies of 90%, 99%, and 99.9% of Airborne Contaminants⁴

Air Changes/Hour (ACH)	Minutes Required for a Removal Efficiency Of:		
	90%	99%	99.9%
1	138	276	414
2	69	138	207
3	46	92	138
4	35	69	104
5	28	55	83
6	23	46	69
7	20	39	59
8	17	35	52
9	15	31	46
10	14	28	41
11	13	25	38
12	12	23	35
13	11	21	32
14	10	20	30
15	9	18	28
16	9	17	26
17	8	16	24
18	8	15	23
19	7	15	22
20	7	14	21
25	6	11	17
30	5	9	14
35	4	8	12
40	3	7	10
45	3	6	9
50	3	6	8

This table has been adapted from the formula for the rate of purging airborne contaminants.¹³¹²

Values have been derived from the formula $t_1 = [\ln (C_2 / C_1) / (Q / V)] \times 60$, with $T_1 = 0$ and

C_2 / C_1 - (removal efficiency / 100), and where:

t_1 = initial timepoint

C_1 = initial concentration of contaminant

C_2 = final concentration of contaminant

Q = air flow rate (cubic feet/hour CFH)

V = room volume (cubic feet)

$Q / V = ACH$

The times given assume perfect mixing of the air within the space (i.e., mixing factor = 1). However, perfect mixing usually does not occur, and the mixing factor could be as high as 10 if air distribution is very poor.²⁰⁹ The required time is derived by multiplying the appropriate time from the table by the mixing factor that has been determined for the booth or room. The factor and required time should be included in the operating instruction is provided by the manufacturer of the booth or enclosure, and these instructions should be followed.

2. Air Sampling for Aerosols Containing Legionellae

Air sampling is an insensitive means of detecting *Legionella pneumophila*, and is of limited practical value in environmental sampling for this pathogen. In certain instances, however, it can be used to: 1) demonstrate the presence of legionellae in aerosol droplets associated with suspected bacterial reservoirs; 2) define the role of certain devices [e.g., showers, faucets, evaporative condensers] in disease transmission; and 3) quantitate and determine the size of the droplets containing legionellae.¹³¹³ Stringent controls and calibration are necessary when sampling is used to determine particle size and numbers of viable bacteria.¹³¹⁴ Samplers should be placed in locations where human exposure to aerosols is anticipated, and investigators should wear an NIOSH-approved respirator if sampling involves exposure to potentially infectious aerosols.

Methods used to sample air for legionellae include impingement in liquid, impaction of solid medium, and sedimentation using settle plates.¹³¹³ The Chemical Corps type all-glass impingers (AGI) with the stem 30 mm from the bottom of the flask have been used successfully to sample for legionellae.¹³¹³ Because of the velocity at which air samples are collected, clumps tend to be fragmented, leading to a more accurate count of bacteria present in the air. The disadvantages of this method are: 1) the velocity of collection tends to destroy some vegetative cells; 2) it does not differentiate particle sizes; and 3) AGIs are easily broken in the field. Yeast extract broth (0.25%) is the recommended liquid medium for AGI sampling of legionellae;¹³¹⁴ standard methods for water samples can be used to culture these samples.

Andersen samplers are viable particle samplers in which particles pass through jet orifices of decreasing size in cascade fashion until they impact on an agar surface.¹¹³¹ The agar plates are then removed and incubated. The stage distribution of the legionellae should indicate the extent to which the bacteria would have penetrated the respiratory system. The advantages of this sampling method are: 1) the equipment is more durable during use; 2) the sampler can determine the number and size of droplets containing legionellae; 3) the agar plates can be placed directly in an incubator with no further manipulations; and 4) both selective and nonselective BCYE agar can be used in an Andersen sampler. If the samples must be shipped to a laboratory, they should be packed and shipped without refrigeration as soon as possible.

3. Calculation of Air Sampling Results¹¹³¹

Assuming that each colony on the agar plate is the growth from a single bacteria-carrying particle, the contamination of the air being sampled is determined from the number of colonies counted. The airborne microorganisms may be reported in terms of the number per cubic foot of air sampled. The following formulas can be applied to convert the colony count to organisms per cubic foot of air sampled:

For solid agar impactor samplers:

$$\frac{C}{R \times P} = N \quad \text{Where } N = \text{number of organisms collected per cubic foot of air sampled}$$

C = total plate count
R = airflow rate in cubic feet per minute
P = duration of sampling period (minutes)

For liquid impingers:

$$\frac{C \times V}{Q \times P \times R} = N \quad \text{Where } C = \text{total number of colonies from all aliquots plated}$$

V = final volume in mL of collecting media
Q = total number of mLs plated
P, R, and N as above

4. Ventilation Specifications for Healthcare Facilities¹²⁰

The following tables from the 2000 draft of the AIA *Guidelines for Design and Construction of Hospital and Health Care Facilities* are reprinted with permission of the publisher (AIA).

Table B.2. Ventilation Requirements for Areas Affecting Patient Care in Hospitals and Outpatient Facilities¹ (Table 7.2 AIA revision 2000)

Area designation	Air movement relationship to adjacent area ²	Minimum air changes of outdoor air per hour ³	Minimum total air changes per hour ^{4,20}	All air exhausted directly to outdoors ⁵	Recirculated by means of room units ⁶	Relative humidity ⁷ (%)	Design temperature ⁸ (degrees F/C)
<u>SURGERY AND CRITICAL CARE</u>							
Operating/surgical cystoscopic rooms ^{9, 15}	Out	3	15	--	No	30-60	68-73 (20-23) ¹⁹
Delivery room ⁹	Out	3	15	--	No	30-60	68-73 (20-23)
Recovery room ⁹	--	2	6	--	No	30-60	70-75 (21-24)
Critical and intensive care	--	2	6	--	No	30-60	70-75 (21-24)
Newborn intensive care	--	2	6	--	No	30-60	72-78 (22-26)
Treatment room ¹⁰	--	--	6	--	--	--	75 (24)
Trauma room ¹⁰	Out	3	15	--	No	30-60	70-75 (21-24)
Anesthesia gas storage	In	--	8	Yes	--	--	--
Endoscopy	In	2	6	--	No	30-60	68-73 (20-23)
Bronchoscopy ¹⁵	In	2	12	Yes	No	30-60	68-73 (20-23)
ER waiting rooms	In	2	12	Yes ¹⁸	--	--	70-75 (21-24)
Radiology waiting rooms	In	2	12	Yes ¹⁸	--	--	70-75 (21-24)
Procedure room	Out	3	15	--	No	30-60	70-75 (21-24)
Rooms with gluteraldehyde ²¹	In	--	15	Yes	No	--	--
<u>NURSING</u>							
Patient room	--	2	6 ¹⁶	--	--	*--	70-75 (21-24)
Toilet room	In	--	10	Yes	--	--	--
Newborn nursery suite	--	2	6	--	No	30-60	72-78 (22-26)
Protective Environment Room ^{11, 15}	Out	2	12	--	No	--	75 (24)
Airborne infection isolation room ^{12, 15}	In	2	12	Yes ¹⁸	No	--	75 (24)
Isolation alcove or anteroom ^{11, 12}	In/Out	--	10	Yes	No	--	--
Labor/delivery/recovery	--	2	6 ¹⁶	--	--	--	70-75 (21-24)
Labor/delivery/recovery/postpartum	--	2	6 ¹⁶	--	--	--	70-75 (21-24)
Patient corridor	--	--	2	--	--	--	--
<u>ANCILLARY</u>							
Radiology ¹³							
X-ray (surgical/critical care and catheterization)	Out	3	15	--	No	30-60	70-75 (21-24)
X-ray (diagnostic & treat.)	--	--	6	--	--	--	75 (24)
Darkroom	In	--	10	Yes	No	--	--

Area designation	Air movement relationship to adjacent area ²	Minimum air changes of outdoor air per hour ³	Minimum total air changes per hour ⁴	All air exhausted directly to outdoors ⁵	Recirculated by means of room units ⁶	Relative humidity ⁷ (%)	Design temperature ⁸ (degrees F/C)
LABORATORY							
General ¹³	--	--	6	--	--	--	75 (24)
Biochemistry ¹³	Out	--	6	--	No	--	75 (24)
Cytology	In	--	6	Yes	No	--	75 (24)
Glass washing	In	--	10	Yes	--	--	--
Histology	In	--	6	Yes	No	--	75 (24)
Microbiology ¹³	In	--	6	Yes	No	--	75 (24)
Nuclear medicine	In	--	6	Yes	No	--	75 (24)
Pathology	In	--	6	Yes	No	--	75 (24)
Serology	Out	--	6	--	No	--	75 (24)
Sterilizing	In	--	10	Yes	--	--	--
Autopsy room ¹⁵	In	--	12	Yes	No	--	--
Nonrefrigerated body-holding room	In	--	10	Yes	--	--	70 (21)
Pharmacy	Out	--	4	--	--	--	--
<u>DIAGNOSTIC AND TREATMENT</u>							
Examination room	--	--	6	--	--	--	75 (24)
Medication room	Out	--	4	--	--	--	--
Treatment room	--	--	6	--	--	--	75 (24)
Physical therapy and hydrotherapy	In	--	6	--	--	--	75 (24)
Soiled workroom or soiled holding	In	--	10	Yes	No	--	--
Clean workroom or clean holding	Out	--	4	--	--	--	--
<u>STERILIZING AND SUPPLY</u>							
ETO-sterilizer room	In	--	10	Yes	No	30-60	75 (24)
Sterilizer equipment room	In	--	10	Yes	--	--	--
Central medical and surgical supply							
Soiled or decontamination room	In	--	6	Yes	No	--	68-73 (20-23)
Clean workroom	Out	--	4	--	No	30-60	75 (24)

Area designation	Air movement relationship to adjacent area ²	Minimum air changes of outdoor air per hour ³	Minimum total air changes per hour ⁴	All air exhausted directly to outdoors ⁵	Recirculated by means of room units ⁶	Relative humidity ⁷ (%)	Design temperature ⁸ (degrees F/C)
Ware washing	In	--	10	Yes	No	--	--
Dietary day storage	In	--	2	--	--	--	--
Laundry, general	--	--	10	Yes	--	--	--
Sterile Storage	Out	--	4	--	--	(Max)70	--
<u>SERVICE</u>							
Food preparation center ¹⁴	--	--	10	--	No	--	--
Soiled linen (sorting and storage)	In	--	10	Yes	No	--	--
Clean linen storage	Out	--	2	--	--	--	--
Soiled linen and trash chute room	In	--	10	Yes	No	--	--
Bedpan room	In	--	10	Yes	--	--	--
Bathroom	In	--	10	--	--	--	75 (24)
Janitor's closet	In	--	10	Yes	No	--	--

Notes

¹The ventilation rates in this table cover ventilation for comfort, as well as for asepsis and odor control in areas of acute care hospitals that directly affect patient care and are determined based on healthcare facilities being predominantly "No Smoking" facilities. Where smoking may be allowed, ventilation rates will need adjustments. Areas where specific ventilation rates are not given in the table shall be ventilated in accordance with ASHRAE Standard 62, *Ventilation for Acceptable Indoor Air Quality*, and ASHRAE *Handbook of Applications*. Specialized patient care areas, including organ transplant units, burn units, specialty procedure rooms, etc., shall have additional ventilation provisions for air quality control as may be appropriate. OSHA standards and/or NIOSH criteria require special ventilation requirements for employee health and safety within healthcare facilities.

²Design of the ventilation system shall provide air movement which is generally from clean to less clean areas. If any form of variable air volume or load shedding system is used for energy conservation, it must not compromise the corridor-to-room pressure balancing relationships or the minimum air changes required by the table.

³To satisfy exhaust needs, replacement air from the outside is necessary. Table 2 does not attempt to describe specific amounts of outside air to be supplied to individual spaces except for certain areas such as those listed. Distribution of the outside air, added to the system to balance required exhaust, shall be as required by good engineering practice. Minimum outside air quantities shall remain constant while the system is in operation.

⁴Number of air changes may be reduced when the room is unoccupied if provisions are made to ensure that the number of air changes indicated is reestablished any time the space is being utilized. Adjustments shall include provisions so that the direction of air movement shall remain the same when the number of air changes is reduced. Areas not indicated as having continuous directional control may have ventilation systems shut down when space is unoccupied and ventilation is not otherwise needed, if the maximum infiltration or exfiltration permitted in Note 2 is not exceeded and if adjacent pressure balancing relationships are not compromised. Air quantity calculations must account for filter loading such that the indicated air change rates are provided up until the time of filter change-out.

⁵Air from areas with contamination and/or odor problems shall be exhausted to the outside and not recirculated to other areas. Note that individual circumstances may require special consideration for air exhaust to the outside, (e.g., in intensive care units in which patients with pulmonary infection are treated) and rooms for burn patients.

*⁶Recirculating room HVAC units refers to those local units that are used primarily for heating and cooling of air, and not disinfection of air. Because of cleaning difficulty and potential for buildup of contamination, recirculating room units shall not be used in areas marked "No." However, for airborne infection control, air may be recirculated within individual isolation rooms if HEPA filters are used. Isolation and intensive care unit rooms may be ventilated by reheat induction units in which only the primary air supplied from a central system passes through the reheat unit. Gravity-type heating or cooling units such as radiators or convectors shall not be used in operating rooms and other special care areas. See Appendix A for a description of recirculation units to be used in isolation rooms.

*⁷The ranges listed are the minimum and maximum limits where control is specifically needed. The maximum and minimum limits are not intended to be independent of a space's associated temperature. The humidity is expected to be at the higher end of the range when the temperature is also at the higher end, and vice versa.

⁸Where temperature ranges are indicated, the systems shall be capable of maintaining the rooms at any point within the range during normal operation. A single figure indicates a heating or cooling capacity of at least the indicated temperature. This is usually applicable when patients may be undressed and require a warmer environment. Nothing in these guidelines shall be construed as precluding the use of temperatures lower than those noted when the patients' comfort and medical conditions make lower temperatures desirable. Unoccupied areas such as storage rooms shall have temperatures appropriate for the function intended.

⁹National Institute for Occupational Safety and Health (NIOSH) Criteria Documents regarding Occupational Exposure to Waste Anesthetic Gases and Vapors, and Control of Occupational Exposure to Nitrous Oxide indicate a need for both local exhaust (scavenging) systems and general ventilation of the areas in which the respective gases are utilized.

¹⁰The term *trauma room* as used here is the operating room space in the emergency department or other trauma reception area that is used for emergency surgery. The first aid room and/or "emergency room" used for initial treatment of accident victims may be ventilated as noted for the "treatment room." Treatment rooms used for Bronchoscopy shall be treated as Bronchoscopy rooms. Treatment rooms used for cryosurgery procedures with nitrous oxide shall contain provisions for exhausting waste gases.

*¹¹ The protective environment airflow design specifications protect the patient from common environmental airborne infectious microbes (i.e., *Aspergillus* spores). These special ventilation areas shall be designed to provide directed airflow from the cleanest patient care area to less clean areas. These rooms shall be protected with HEPA filters at 99.97 percent efficiency for a 0.3 μm sized particle in the supply airstream. These interrupting filters protect patient rooms from maintenance-derived release of environmental microbes from the ventilation system components. Recirculation HEPA filters can be used to increase the equivalent room air exchanges. Constant volume airflow is required for consistent ventilation for the protected environment. If the facility determines that airborne infection isolation is necessary for protective environment patients, an anteroom

should be provided. Rooms with reversible airflow provisions for the purpose of switching between protective environment and airborne infection isolation functions are not acceptable.

¹²The infectious disease isolation room described in these guidelines is to be used for isolating the airborne spread of infectious diseases, such as measles, varicella, or tuberculosis. The design of airborne infection isolation (AII) rooms should include the provision for normal patient care during periods not requiring isolation precautions. Supplemental recirculating devices may be used in the patient room, to increase the equivalent room air exchanges; however, such recirculating devices do not provide the outside air requirements. Air may be recirculated within individual isolation rooms if HEPA filters are used. Rooms with reversible airflow provisions for the purpose of switching between protective environment and AII functions are not acceptable.

¹³When required, appropriate hoods and exhaust devices for the removal of noxious gases or chemical vapors shall be provided (see Section 7.31.D1.n and o and NFPA 99).

¹⁴Food preparation centers shall have ventilation systems whose air supply mechanisms are interfaced appropriately with exhaust hood controls or relief vents so that exfiltration or

infiltration to or from exit corridors does not compromise the exit corridor restrictions of NFPA 90A, the pressure requirements of NFPA 96, or the maximum defined in the table. The number of air changes may be reduced or varied to any extent required for odor control when the space is not in use. See Section 7.31.D1.p.

^{*15}Differential pressure shall be a minimum of 0.01” water gauge (2.5 Pa). If alarms are installed, allowances shall be made to prevent nuisance alarms of monitoring devices.

¹⁶Total air changes per room for patient rooms, labor/delivery/recovery rooms, and labor/delivery/recovery/postpartum rooms may be reduced to 4 when supplemental heating and/or cooling systems (radiant heating and cooling, baseboard heating, etc). are used.

¹⁷ Not used.

¹⁸ If it is not practical to exhaust the air from the airborne infection isolation room to the outside, the air may be returned through HEPA filters to the air-handling system exclusively serving the isolation room.

¹⁹Some surgeons may require room temperatures which are outside of the indicated range. All operating room design conditions shall be developed in consultation with surgeons, anesthesiologists, and nursing staff.

²⁰Air change requirements indicated are minimum values. Higher values should be used when required to maintain indicated room conditions (temperature and humidity), based on the cooling load of the space (lights, equipment, people, exterior walls and windows, etc.).

^{A6}Recirculating devices with HEPA filters may have potential uses in existing facilities as interim, supplemental environmental controls to meet requirements for the control of airborne infectious agents. Limitations in design must be recognized. The design of either portable or fixed systems should prevent stagnation and short circuiting of airflow. The supply and exhaust locations should direct clean air to areas where health care workers are likely to work, across the infectious source, and then to the exhaust, so that the health care worker is not in position between the infectious source and the exhaust location. The design of such systems should also allow for easy access for scheduled preventative maintenance and cleaning.

^{A7}Recirculating devices with HEPA filters may have potential uses in existing facilities as interim, supplemental environmental controls to meet requirements for the control of airborne infectious agents. Limitations in design must be recognized. The design of either portable or fixed systems should prevent stagnation and short circuiting of airflow. The supply and exhaust locations should direct clean air to areas where health care workers are likely to work, across the infectious source, and then to the exhaust, so that the health care worker is not in position between the infectious source and the exhaust location. The design of such systems should also allow for easy access for scheduled preventative maintenance and cleaning.

^{A11}The verification of airflow direction can include a simple visual method such as smoke trail, ball-in-tube, or flutterstrip. These devices will require a minimum differential air pressure to indicate airflow direction.

^{A15}The verification of airflow direction can include a simple visual method such as: smoke trail, ball-in-tube, or flutterstrip. These devices will require a minimum differential air pressure to indicate airflow direction.

Table B.3. Filter Efficiencies for Central Ventilation and Air Conditioning Systems in General Hospitals (Table 7.3 AIA revision 2000)

Area designation	No. filter beds	Filter bed no. 1 (%)	Filter bed no. 2 (%)
All areas for inpatient care, treatment, and diagnosis, and those areas providing direct service or clean supplies such as sterile and clean processing, etc.	2	30	90
Protective environment room	2	30	99.97
Laboratories	1	80	--
Administrative, bulk storage, soiled holding areas, food preparation areas, and laundries	1	30	--

Notes. Additional roughing or prefilters should be considered to reduce maintenance required for filters with efficiency higher than 75 percent. The filtration efficiency ratings are based on average dust spot efficiency per ASHRAE 52.1-92.

Table B.4. Filter Efficiencies for Central Ventilation and Air Conditioning Systems in Outpatient Facilities (Table 9.1 AIA revision 2000)

Area designation	No. filter beds	Filter bed no. 1	Filter bed no. 2
All areas for patient care, treatment, and/or diagnosis, and those areas providing direct service or clean supplies such as sterile and clean processing, etc. ¹	2	30	90
Laboratories	1	80	--
Administrative, bulk storage, soiled holding areas, food preparation areas, and laundries	1	30	--

Notes. Additional roughing or prefilters should be considered to reduce maintenance required for main filters. The filtration efficiency ratings are based on dust spot efficiency per ASHRAE 59-92.

¹These requirements do not apply to small primary (neighborhood) outpatient facilities or outpatient facilities that do not perform invasive applications or procedures.

Table B.5. Pressure Relationships and Ventilation of Certain Areas of Nursing Facilities¹ (Table 8.1 AIA revision 2000)

Area designation	Air movement relationship to adjacent area ²	Minimum air changes of outdoor air per hour ³	Minimum total air changes per hour ⁴	All air exhausted directly to outdoors ⁵	Recirculated by means of room units ⁶	Relative humidity ⁷ (%)	Design temperature ⁸ (degrees F/C)
Resident room	--	2	2	--	--	⁹	70-75 (21-24)
Resident unit corridor	--	--	4	--	--	⁹	--
Resident gathering areas		4	4				
Toilet Room	In	--	10	Yes	No	--	--
Dining rooms	--	2	4	--	--		75
Activity rooms, if provided	--	4	4	--	--	--	--
Physical therapy	In	2	6	--	--	--	75 (24)
Occupational therapy	In	2	6	--	--	--	75 (24)
Soiled workroom or soiled holding	In	2	10	Yes	No	--	--
Clean workroom or clean holding	Out	2	4	--	--	(Max) 70	75 (24)
Sterilizer exhaust room	In	--	10	Yes	No	--	--
Linen and trash chute room, if provided	In	--	10	Yes	No	--	--
Laundry, general, if provided	--	2	10	Yes	No	--	--
Soiled linen sorting and storage	In	--	10	Yes	No	--	--
Clean linen storage	Out	--	2	Yes	No	--	--
Food preparation facilities ¹²	--	2	10	Yes	Yes	--	--
Dietary warewashing	In	--	10	Yes	Yes	--	--
Dietary storage areas	--	--	2	Yes	No	--	--
Housekeeping rooms	In	--	10	Yes	No	--	--
Bathing rooms	In	--	10	Yes	No	--	75 (24)

Notes

¹The ventilation rates in this table cover ventilation for comfort, as well as for asepsis and odor control in areas of nursing facilities that directly affect resident care and are determined based on nursing facilities being predominantly "No Smoking" facilities. Where smoking may be allowed, ventilation rates will need adjustments. Areas where specific ventilation rates are not given in the table shall be ventilated in accordance with ASHRAE Standard 62, *Ventilation for Acceptable Indoor Air Quality*, and ASHRAE *Handbook of Applications*. OSHA standards and/or NIOSH criteria require special ventilation requirements for employee health and safety within nursing facilities.

²Design of the ventilation system shall, insofar as possible, provide that air movement is from "clean to less clean" areas. However, continuous compliance may be impractical with full utilization of some forms of variable air volume and load shedding systems that may be used for energy conservation. Areas that do require positive and continuous control are noted with "Out" or "In" to indicate the required direction of air movement in relation to the space named. Rate of air movement may, of course, be varied as needed within the limits required for positive control. Where indication of air movement direction is enclosed in parentheses, continuous directional control is required only when the specialized equipment or device is in use or where room use may otherwise compromise the intent of movement from clean to less clean. Air movement for rooms with dashes and nonpatient areas may vary as necessary to satisfy the requirements of those spaces. Additional adjustments may be needed when space is unused or unoccupied and air systems are deenergized or reduced.

³To satisfy exhaust needs, replacement air from outside is necessary. Table 6 does not attempt to describe specific amounts of outside air to be supplied to individual spaces except for certain areas such as those listed. Distribution of the outside air, added to the system to balance required exhaust, shall be as required by good engineering practice.

⁴Number of air changes may be reduced when the room is unoccupied if provisions are made to ensure that the number of air changes indicated is reestablished any time the space is being utilized. Adjustments shall include provisions so that the direction of air movement shall remain the same when the number of air changes is reduced. Areas not indicated as having continuous directional control may have ventilation systems shut down when space is unoccupied and ventilation is not otherwise needed.

⁵Air from areas with contamination and/or odor problems shall be exhausted to the outside and not recirculated to other areas. Note that individual circumstances may require special consideration for air exhaust to outside.

⁶Because of cleaning difficulty and potential for buildup of contamination, recirculating room units shall not be used in areas marked "No." Isolation rooms may be ventilated by reheat induction units in which only the primary air supplied from a central system passes through the reheat unit. Gravity-type heating or cooling units such as radiators or convectors shall not be used in special care areas.

^{*7}The ranges listed are the minimum and maximum limits where control is specifically needed. See A8.31.D for additional information.

⁸Where temperature ranges are indicated, the systems shall be capable of maintaining the rooms at any point within the range. A single figure indicates a heating or cooling capacity of at least the indicated temperature. This is usually applicable where residents may be undressed and require a warmer environment. Nothing in these guidelines shall be construed as precluding the use of temperatures lower than those noted when the residents' comfort and medical conditions make lower temperatures desirable. Unoccupied areas such as storage rooms shall have temperatures appropriate for the function intended.

^{*9}See A8.31.D1.

^{*10}The protective environment airflow design specifications protect the patient from common environmental airborne infectious microbes (i.e., Aspergillus spores). These special ventilation areas shall be designed to provide directed airflow from the cleanest patient care area to less clean areas. These rooms shall be protected with HEPA filters at 99.97 percent efficiency for a 0.3 μm sized particle in the supply airstream. These interrupting filters protect patient rooms from maintenance-derived release of environmental microbes from the ventilation system components. Recirculation HEPA filters can be used to increase the equivalent room air exchanges. Constant volume airflow is required for consistent ventilation for the protected environment. If the facility determines that airborne infection isolation is necessary for protective environment patients, an anteroom shall be provided. Rooms with reversible airflow provisions for the purpose of switching between protective isolation and airborne infection isolation functions are not acceptable.

¹¹The infectious disease isolation room described in these guidelines is to be used for isolating the airborne spread of infectious diseases, such as measles, varicella, or tuberculosis. The design of airborne infection isolation (AII) rooms should include the provision for normal patient care during periods not requiring isolation precautions. Supplemental recirculating devices may be used in the patient room, to increase the equivalent room air exchanges; however, such recirculating devices do not provide the outside air requirements.

Air may be recirculated within individual isolation rooms if HEPA filters are used. Rooms with reversible airflow provisions for the purpose of switching between protective isolation and airborne infection isolation functions are not acceptable.

¹²Food preparation facilities shall have ventilation systems whose air supply mechanisms are interfaced appropriately with exhaust hood controls or relief vents so that exfiltration or infiltration to or from exit corridors does not compromise the exit corridor restrictions of NFPA 90A, the pressure requirements of NFPA 96, or the maximum defined in the table. The number of air changes may be reduced or varied to any extent required for odor control when the space is not in use.

^{A10} Recirculating devices with HEPA filters may have potential uses in existing facilities as interim, supplemental environmental controls to meet requirements for the control of airborne infectious agents. Limitations in design must be recognized. The design of either portable or fixed systems should prevent stagnation and short circuiting of airflow. The supply and exhaust locations should direct clean air to areas where health care workers are likely to work, across the infectious source, and then to the exhaust, so that the health care worker is not in position between the infectious source and the exhaust location. The design of such systems should also allow for easy access for scheduled preventative maintenance and cleaning.

Table B.6. Filter Efficiencies for Central Ventilation and Air Conditioning Systems in Nursing Facilities (Table 8.2 AIA revision 2000)

Area Designation	Minimum number of filter beds	Filter efficiencies (%)	
		Filter bed no. 1	Filter bed no. 2
All areas for inpatient care, treatment, and/or diagnosis, and those areas providing direct service or clean supplies	2	30	80
Administrative, bulk storage, soiled holding, laundries, food preparation areas	1	30	

Note: The filtration efficiency ratings are based on average dust spot efficiency per ASHRAE 52.1-92.

B.7. Filter Efficiencies for Central Ventilation and Air Conditioning Systems in Psychiatric Hospitals (Table 11.1 AIA revision 2000)

Area	Minimum number of filter beds	Filter efficiencies (%)	
		Filter bed 1	Filter bed 2
All areas for inpatient care, treatment, and diagnosis, and those areas providing direct services	2	30	90
Administrative, bulk storage, soiled holding, laundries, food preparation areas	1	30	-

Note. Filtration efficiency ratings are based on dust spot efficiency per ASHRAE 52-92.

Appendix C - Water

1. Biofilms

Microorganisms have a tendency to associate with and stick to surfaces. These adherent organisms can initiate and develop biofilms, which are comprised of cells embedded in a matrix of extracellularly produced polymers and associated abiotic particles.¹³¹⁵ It is inevitable that biofilms will form in most water systems, and in the healthcare facility environment, biofilms may be found in the potable water supply piping, hot water tanks, air conditioning cooling towers, or in sinks, sink traps, aerators, or shower heads. Biofilms, especially in water systems, are not present as a continuous slime or film, but are more often scanty and heterogeneous in nature.¹³¹⁶ Biofilms may form under stagnant as well as flowing conditions, so storage tanks, in addition to water system piping, may be vulnerable to the development of biofilm, especially if water temperatures are low enough to allow the growth of thermophilic bacteria such as *Legionella* spp. and if these structures and equipment are not cleaned for extended periods of time.¹³¹⁷

Algae, protozoa, and fungi may be present in biofilms, but the predominant microorganisms of water system biofilms are gram negative bacteria. Although most of these organisms will not normally pose a problem for healthy individuals, certain biofilm bacteria (e.g., *Pseudomonas aeruginosa*, *Klebsiella* spp., *Pantoea agglomerans*, *Enterobacter cloacae*) all may be agents for opportunistic infections for immunocompromised individuals.^{1318, 1319} These biofilm organisms may easily contaminate indwelling medical devices or intravenous (IV) fluids, and could be transferred on the hands of health care workers.^{1318 - 1321} Biofilms may potentially provide an environment for the survival of pathogenic organisms, such as *Legionella pneumophila* and *E. coli* O157:H7. Although the association of biofilms and medical devices provides a plausible explanation for a variety of healthcare-associated infections, it is not clear how the presence of biofilms in the water system may influence the rates of healthcare-associated waterborne infection.

Organisms within biofilms behave quite differently than their planktonic (free floating) counterparts. Research has shown that biofilm-associated organisms are more resistant to antibiotics and disinfectants than are planktonic organisms, either because the cells are protected by the polymer matrix, or because they are physiologically different.^{1322 - 1327} Nevertheless, municipal water utilities attempt to maintain a chlorine residual in the distribution system to discourage microbiological growth. Though chlorine in its various forms is a proven disinfectant, it has been shown to be less effective against biofilm bacteria.¹³²⁵ Higher levels of chlorine for longer contact times are necessary to eliminate biofilms.

Routine sampling of healthcare facility water systems for biofilms is not warranted. If an epidemiologic investigation points to the water supply system as a possible source of infection, then water sampling for biofilm organisms should be considered so that prevention and control strategies can be developed. Once a biofilm is established, it is difficult to totally remove in existing piping. Strategies to remediate biofilms in a water system would include flushing the system piping, hot water tank, dead legs, and those areas of the facility's water system subject to low or intermittent flow. The benefits of this treatment would include: 1) elimination of corrosion deposits and sludge from the bottom of hot water tanks; 2) removal of biofilms from shower heads and sink aerators; and 3) circulation of fresh water containing elevated chlorine residuals into the healthcare facility water system.

The general strategy for evaluating water system biofilm depends on a comparison of the bacteriological quality of the incoming municipal water and that of water sampled from within facility's distribution system. Heterotrophic plate counts and coliform counts, both of which are routinely run by the municipal water utility, will at least provide in indication of the potential for biofilm formation. Heterotrophic plate count levels in potable water should be <500 CFU/mL. These levels may increase on occasion, but counts consistently >500 CFU/mL would indicate a general decrease in water quality. A direct correlation between heterotrophic plate count and biofilm levels has been demonstrated.¹³²⁷ Therefore, an increase in heterotrophic plate count would suggest a greater rate and extent of biofilm formation in a healthcare facility water system. The water supplied to the facility should also contain <1 coliform bacteria/100 mL. Coliform bacteria are indicator organisms whose presence in the distribution system could indicate the presence of

fecal contamination. It has been shown that coliform bacteria can colonize biofilms within drinking water systems, hence intermittent contamination of a water system with these organisms could lead to colonization of the system.

Water samples can be collected from throughout the healthcare facility system, including both hot and cold water sources; samples should be cultured by standard methods.⁸⁹⁵ If heterotrophic plate counts in the facility water system are higher than those at the point of water entry to the building, it can be concluded that the facility water quality has diminished. If biofilms are detected in the facility water system and determined by an epidemiologic and environmental investigation to be a reservoir for healthcare-associated pathogens, the municipal water supplier could be contacted with a request to provide higher chlorine residuals in the distribution system, or the healthcare facility could consider installing a supplemental chlorination system.

Sample collection sites for biofilm in healthcare facilities include hot water tanks, and shower heads and faucet aerators, especially in immunocompromised patient-care areas. Swabs should be placed into tubes containing phosphate buffered water, pH 7.2 or phosphate buffered saline, shipped to the laboratory under refrigeration and processed within 24 hrs. of collection by vortexing with glass beads and plated onto a nonselective medium (e.g., Plate Count Agar or R2A medium) and selective media (e.g., media for *Legionella* spp. isolation) after serial dilution. If the plate counts are elevated above levels in the water (i.e. comparing the plate count per square centimeter of swabbed surface to the plate count per milliliter of water), then biofilm formation can be suspected. In the case of an outbreak, it would be advisable to isolate organisms from these plates to determine whether the suspect organisms are present in the biofilm or water samples and compare them to the organisms isolated from patient specimens.

2. Water and Dialysate Sampling Strategies in Dialysis

In order to detect the low total viable heterotrophic plate counts outlined by the current AAMI standards for water and dialysate in dialysis settings, it is necessary to use standard quantitative culture techniques with appropriate sensitivity levels.^{792, 793} The membrane filter technique is particularly suited for this application because it permits large volumes of water to be assayed.⁷⁹⁴ Since the membrane filter technique may not be readily available in clinical laboratories, the spread plate assay can be used as an alternative.⁷⁹⁴ If the spread plate assay is used, however, the standard prohibits the use of a calibrated loop when applying sample to the plate. The prohibition is based on the low sensitivity of the calibrated loop. A standard calibrated loop transfers 0.001 mL of sample to the culture medium, so that the minimum sensitivity of the assay is 1,000 CFU/mL. This level of sensitivity is unacceptable when the maximum allowable limit for microorganisms is 200 CFU/mL. Therefore, when the spread plate method is used, a pipette must be used to place 0.1 to 0.5 mL of water on the culture medium.

The current AAMI Standard specifically prohibits the use of nutrient-rich media (e.g., blood agar, chocolate agar) in dialysis water and dialysate assays because these culture media are too rich for growth of the naturally occurring organisms found in water. Debate continues within AAMI, however, as to the most appropriate culture medium and incubation conditions to be used. The original clinical observations on which the microbiological requirements of this standard were based used Standard Methods Agar (SMA), a medium containing relatively few nutrients.⁶⁴² The use of Tryptic Soy Agar (TSA), a general purpose medium for isolating and cultivating microorganisms was recommended in later versions of the standard because it was thought more appropriate for culturing bicarbonate-containing dialysate.^{749, 750, 795} Moreover, culturing systems based on TSA are readily available from commercial sources. Several studies, however, have shown that the use of nutrient-poor media, such as R2A, results in an increased recovery of bacteria from water.^{1328, 1329} The original standard also specified incubation for 48 hours at 35°C - 37°C (95°F - 98.6°F) before enumeration of bacterial colonies. Extending the culturing time up to 168 hours, or 7 days and using incubation temperatures of 23°C - 28°C (73.4°F - 82.4°F) have also been shown to increase the recovery of bacteria.^{1328, 1329} Other investigators, however, have not found such clear cut differences between culturing techniques.^{795, 1330} After considerable discussion, the AAMI Committee has not reached a consensus regarding changes in the assay technique, and the use of TSA for 48 hours at 37°C (98.6°F) remains the recommended method. It should be recognized, however, that these culturing conditions may underestimate the bacterial burden in the water and fail to identify the presence of some organisms.

Specifically, the recommended method may not detect the presence of various NTM that have been associated with several outbreaks of infection in dialysis units.^{31, 32} In these instances, however, the high numbers of mycobacteria in the water were related to the total heterotrophic plate counts, each of which was significantly greater than that allowable by the AAMI Standard.

Endotoxin can be tested by one of two types of assays - kinetic (colorimetric or turbidimetric) or gel-clot. Endotoxin units are assayed by the *Limulus* Amebocyte Lysate (LAL) method. Because endotoxins differ in their activity on a mass basis, their activity is referred to a standard *Escherichia coli* endotoxin. The current standard (EC-6) is prepared from *E. coli* O113:H10. The relationship between mass of endotoxin and its activity varies with both the lot of LAL and the lot of control standard endotoxin used. Since standards for endotoxin were harmonized in 1983 with the introduction of EC-5, the relationship between mass and activity of endotoxin has been approximately 5 - 10 EU/ng. Studies to harmonize standards have led to the measurement of endotoxin units (EU) where 5 EU is equivalent to 1 ng *E. coli* O55:B5 endotoxin.¹³³¹

In summary, water used to prepare dialysate and to reprocess hemodialyzers should not contain a total microbial count >200 CFU/mL as determined by assay on TSA agar for 48 hrs. at 36°C (96.8°F), and no more than 5 endotoxin units (EU) per mL. Currently the EU standard applies only to the water used to reprocess the hemodialyzers. The dialysate at the end of a dialysis treatment should not contain >2,000 CFU/mL.^{31, 32, 644, 750}

3. Water Sampling Strategies and Culture Techniques for Detecting Legionellae

Legionella spp. are ubiquitous and can be isolated from 20% - 40% of freshwater environments, including man-made water systems.^{1332, 1333} In healthcare facilities, however, it is difficult to determine a course of remedial action based on the presence of legionellae in potable water since it appears that their presence rarely results in disease among immunocompetent patients.

The issue of regularly scheduled microbiological monitoring for legionellae remains controversial, as detection of legionellae in an environmental source is not necessarily evidence of the potential for disease.¹³³⁴

There is general agreement that monitoring is warranted in order to identify the source of an outbreak of legionellosis or to evaluate the efficacy of biocides or prevention measures. CDC recommends aggressive maintenance/disinfection protocols for devices known to transmit legionellae but does not recommend regularly scheduled microbiologic assays for the bacteria.⁴⁰⁵ Monitoring the water, however, may be considered in special settings where people are highly susceptible to illness and mortality due to *Legionella* infection (e.g., HSCT units, organ transplant units) within a hospital.⁹ In the absence of associated disease, however, there is no clear evidence that basing interventions on microbiologic assays will lead to a reduction in Legionnaires' disease cases or outbreaks.

Examination of water samples is the most efficient microbiologic method for identifying sources of legionellae, which is an integral part of an epidemiologic investigation into healthcare-associated Legionnaires' disease. Because of the diversity of plumbing and HVAC systems in healthcare facilities, the number and types of sites that should be tested must be determined on an individual basis prior to collection of water samples. A previously published environmental sampling protocol that addressed sampling site selection in hospitals can serve as a prototype for sampling in other institutions.¹¹²² Any water source that may be aerosolized should be considered a potential source for the transmission of legionellae. The bacteria are rarely found in municipal water supplies and tend to colonize plumbing systems and point-of-use devices. To colonize a system the bacteria must multiply and this requires temperatures >25°C (>77°F).¹³³⁵ Therefore, legionellae are most commonly found in hot water systems. The bacteria do not survive drying, so condensate from air-conditioning equipment, which frequently evaporates, is not a likely source.¹³³⁶

Water samples and swabs of point-of-use devices or system surfaces should be collected when sampling for legionellae.¹³¹⁴ Swabs of system surfaces (Table C.1) allow sampling of biofilms, which frequently contain legionellae. When culturing faucet aerators and shower heads, swabs of these surfaces should be collected first; water samples are then collected from these fixtures after the aerators or shower heads are removed.

Collection and culture techniques are outlined in Table C.2. Swabs can be streaked directly onto agar plates if those are readily available at the collection site. If the swabs and water samples must be transported back to a laboratory for processing, immersing the individual swabs in sample water minimizes drying during transit. Place swabs and water samples in insulated coolers to protect the specimens from temperature extremes.

Table C.1. Possible Sampling Sites for *Legionella* spp. in Healthcare Facilities¹¹²²

-
- Potable water system:
 - incoming water main
 - water softener
 - holding tanks/cisterns
 - water heater tanks (at the inflow and outflow sites)
 - Potable water outlets, especially those located in or near case-patients' rooms
 - faucets or taps
 - showers
 - Cooling tower/evaporative condenser
 - Make-up water (e.g., water added to the system to replace that lost by evaporation, drift, leakage)
 - Basin (e.g., area under the tower for collection of cooled water)
 - Sump (e.g., section of basin from which cooled water returns to heat source)
 - Heat source (e.g., chillers)
 - Humidifiers (e.g., nebulizers)
 - Bubblers for oxygen
 - Water used for respiratory therapy equipment
 - Other sources
 - Decorative fountains
 - Irrigation equipment
 - Fire/sprinkler system (if recently used)
 - Whirlpools, spas
-

Table C.2. Procedure for Collecting and Processing Environmental Specimens for *Legionella* spp.^{1122, 1314}

-
1. Collect water (if possible, 1-L samples) in sterile, screw-top bottles.
 2. Collect culture-swabs of the internal surfaces of faucets, aerators, and shower heads; in a sterile, screw-top container, such as a 50-mL plastic centrifuge tube, submerge each swab in 5-10 mL of sample water taken from the same device from which the sample was obtained.
 3. As soon as possible after collection, water samples and swabs should be transported to and processed in a laboratory proficient at culturing water specimens for *Legionella* spp. Samples may be transported at room temperature but must be protected from temperature extremes. Samples that are not processed within 24 hours of collection should be refrigerated.
 4. Test samples for the presence of *Legionella* spp. by using semi-selective culture media using procedures specific for the cultivation and detection of *Legionella* spp. (Detection of *Legionella* spp. antigen by the direct fluorescent antibody technique is not suitable for environmental samples.^{1337 - 1339} In addition, the use of polymerase chain reaction for identification of *Legionella* spp. is not recommended until more data regarding the sensitivity and specificity of this procedure are available).¹³⁴⁰
-

4. Procedure for Cleaning Cooling Towers and Related Equipment

- I. Before chemical disinfection and mechanical cleaning:
 - A. Provide protective equipment to workers who perform the disinfection, to prevent their exposure to a) chemicals used for disinfection and b) aerosolized water containing *Legionella* sp. Protective equipment may include full-length protective clothing, boots, gloves, goggles, and a full-

or half-face mask that combines a HEPA filter and chemical cartridges to protect against airborne chlorine levels of up to 10 mg/L.

B. Shut off cooling-tower.

1. If possible, shut off the heat source.
2. Shut off fans, if present, on the cooling tower/evaporative condenser (CT/EC).
3. Shut off the system blowdown (i.e., purge) valve. Shut off the automated blowdown controller, if present, and set the system controller to manual.
4. Keep make-up water valves open.
5. Close building air-intake vents within at least 30 m of the CT/EC until after the cleaning procedure is complete.
6. Continue operating pumps for water circulation through the CT/EC.

II. Chemical disinfection

A. Add fast-release, chlorine-containing disinfectant in pellet, granular, or liquid form, and follow safety instructions on the product label. Examples of disinfectants include sodium hypochlorite (NaOCl) or calcium hypochlorite (Ca[OCl]₂), calculated to achieve initial free residual chlorine (FRC) of 50 mg/L: either 3.0 lbs (1.4 kg) industrial grade NaOCl (12% - 15% available Cl) per 1,000 gal of CT/EC water; 10.5 lbs (4.8 kg) domestic grade NaOCl (3% - 5% available Cl) per 1,000 gal of CT/EC water; or 0.6 lb (0.3 kg) Ca[OCl]₂ per 1,000 gal of CT/EC water. If significant biodeposits are present, additional chlorine may be required. If the volume of water in CT/EC is unknown, it can be estimated (in gallons) by multiplying either the recirculation rate in gallons per minute by 10 or the refrigeration capacity in tons by 30. Other appropriate compounds may be suggested by a water-treatment specialist.

B. Record the type and quality of all chemicals used for disinfection, the exact time the chemicals were added to the system, and the time and results of FRC and pH measurements.

C. Add dispersant simultaneously with or within 15 minutes of adding disinfectant. The dispersant is best added by first dissolving it in water and adding the solution to a turbulent zone in the water system. Automatic-dishwasher compounds are examples of low or nonfoaming, silicate-based dispersants. Dispersants are added at 10 - 25 lbs (4.5 -11.25 kg) per 1,000 gal of CT/EC water.

D. After adding disinfectant and dispersant, continue circulating the water through the system. Monitor the FRC by using an FRC-measuring device with the DPD method (e.g., a swimming-pool test kit), and measure the pH with a pH meter every 15 minutes for 2 hours. Add chlorine as needed to maintain the FRC at ≥ 10 mg/L. Because the biocidal effect of chlorine is reduced at a higher pH, adjust the pH to 7.5 - 8.0. The pH may be lowered by using any acid (e.g., muriatic acid or sulfuric acid used for maintenance of swimming pools) that is compatible with the treatment chemicals.

E. Two hours after adding disinfectant and dispersant or after the FRC level is stable at ≥ 10 mg/L, monitor at 2-hour intervals and maintain the FRC at ≥ 10 mg/L for 24 hours.

F. After the FRC level has been maintained at ≥ 10 mg/L for 24 hours, drain the system. CT/EC water may be drained safely into the sanitary sewer. Municipal water and sewerage authorities should be contacted regarding local regulations. If a sanitary sewer is not available, consult local or state authorities (e.g., Department of Natural Resources) regarding disposal of water. If necessary, the drain-off may be dechlorinated by dissipation or chemical neutralization with sodium bisulfite.

G. Refill the system with water and repeat the procedure outlined in steps 2-6 in Section I-B above.

III. Mechanical cleaning

A. After water from the second chemical disinfection has been drained, shut down the CT/EC.

B. Inspect all water-contact areas for sediment, sludge, and scale. Using brushes and/or a low-pressure water hose, thoroughly clean all CT/EC water-contact areas, including the basin, sump, fill, spray nozzles, and fittings. Replace components as needed.

C. If possible, clean CT/EC water-contact areas within the chillers.

IV. After mechanical cleaning

A. Fill the system with water and add chlorine to achieve FRC level of 10 mg/L.

B. Circulate the water for 1 hour, then open the blowdown valve and flush the entire system until the water is free of turbidity.

C. Drain the system.

- D. Open any air-intake vents that were closed before cleaning.
- E. Fill the system with water. CT/EC may be put back into service using an effective water-treatment program.

5. Maintenance Procedures Used to Decrease Survival and Multiplication of *Legionella* spp. in Potable-Water Distribution Systems

1. Provide water at $\geq 50^{\circ}\text{C}$ (122°F) at all points in the heated water system, including the taps. This requires that water in calorifiers (water heaters) be maintained at $\geq 60^{\circ}\text{C}$ (140°F). In the United Kingdom, where maintenance of water temperatures at $\geq 50^{\circ}\text{C}$ (122°F) in hospitals has been mandated, installation of blending or mixing valves at or near taps to reduce the water temperature to $\leq 43^{\circ}\text{C}$ ($\leq 109.4^{\circ}\text{F}$) has been recommended in certain settings to reduce the risk for scald injury to patients, visitors, and health care workers.⁶⁹² However, *Legionella* spp. can multiply even in short segments of pipe containing water at this temperature. Increasing the flow rate from the hot-water-circulation system may help lessen the likelihood of water stagnation and cooling.^{679, 1341} Insulation of plumbing to ensure delivery of cold ($< 20^{\circ}\text{C}$ [$< 68^{\circ}\text{F}$]) water to water heaters (and to cold-water outlets) may diminish the opportunity for bacterial multiplication.³⁹⁷ Both dead legs and capped spurs within the plumbing system provide areas of stagnation and cooling to $< 50^{\circ}\text{C}$ ($< 122^{\circ}\text{F}$) regardless of the circulating water temperature; these segments may need to be removed to prevent colonization.⁶⁷² Rubber fittings within plumbing systems have been associated with persistent colonization, and replacement of these fittings may be required for *Legionella* spp. eradication.¹³⁴²

2. Continuous chlorination to maintain concentrations of free residual chlorine at 1-2 mg/L at the tap. This requires the placement of flow-adjusted, continuous injectors of chlorine throughout the water distribution system. The adverse effects of continuous chlorination include accelerated corrosion of plumbing, which results in system leaks and production of potentially carcinogenic trihalomethanes. However, when levels of free residual chlorine are below 3 mg/L, trihalomethane levels are kept below the maximum safety level recommended by the EPA.^{693, 1343, 1344}

Appendix D - Insects and Microorganisms

Table D.1. Microorganisms Isolated from Arthropods in Healthcare Settings

Insect	Microorganism Category	Microorganisms	References
Cockroaches	Gram-negative bacteria	<i>Acinetobacter</i> spp.; <i>Citrobacter freundii</i> ; <i>Enterobacter</i> spp., <i>E. cloacae</i> ; <i>Escherichia coli</i> ; <i>Flavobacter</i> spp.; <i>Klebsiella</i> spp.; <i>Proteus</i> spp.; <i>Pseudomonas</i> spp., <i>P. aeruginosa</i> , <i>P. fluorescens</i> , <i>P. putida</i> ; <i>Salmonella</i> spp.; <i>Serratia</i> spp., <i>S. marcescens</i> ; <i>Shigella boydii</i>	984, 987, 992, 994, 995, 998
	Gram-positive bacteria	<i>Bacillus</i> spp.; <i>Enterococcus faecalis</i> ; <i>Micrococcus</i> spp.; <i>Staphylococcus aureus</i> , <i>S. epidermidis</i> ; <i>Streptococcus</i> spp., <i>S. viridans</i>	992, 994, 995
	Acid-fast bacteria	<i>Mycobacterium tuberculosis</i>	1000
	Fungi	<i>Aspergillus niger</i> ; <i>Mucor</i> spp.; <i>Rhizopus</i> spp.	988, 995
	Parasites	<i>Endolimax nana</i> ; <i>Entamoeba coli</i>	995
Houseflies	Gram-negative bacteria	<i>Acinetobacter</i> spp.; <i>Campylobacter fetus</i> subsp. <i>jejuni</i> ; <i>Chlamydia</i> spp.; <i>Citrobacter freundii</i> ; <i>Enterobacter</i> spp.; <i>Escherichia coli</i> ; <i>Helicobacter pylori</i> ; <i>Klebsiella</i> spp.; <i>Proteus</i> spp.; <i>Pseudomonas aeruginosa</i> ; <i>Serratia marcescens</i> ; <i>Shigella</i> spp.	983, 984, 986, 989 - 991, 996
	Gram-positive bacteria	<i>Bacillus</i> spp.; <i>Enterococcus faecalis</i> ; <i>Micrococcus</i> spp.; <i>Staphylococcus</i> spp. (coagulase-negative), <i>S. aureus</i> ; <i>Streptococcus</i> spp., <i>S. epidermidis</i> , <i>S. viridans</i>	984, 996
	Fungi / yeasts	<i>Candida</i> spp.; <i>Geotrichum</i> spp.	996
	Parasites	<i>Endolimax nana</i> ; <i>Entamoeba coli</i>	996
	Viruses	Rotavirus	985
Ants	Gram-negative bacteria	<i>Acinetobacter anitratus</i> ; <i>Escherichia coli</i> ; <i>Klebsiella</i> spp.; <i>Neisseria sicca</i> ; <i>Proteus</i> spp.; <i>Providencia</i> spp.; <i>Pseudomonas aeruginosa</i> , <i>P. fluorescens</i>	993
	Gram-positive bacteria	<i>Bacillus</i> spp. <i>B. cereus</i> , <i>B. pumilis</i> ; <i>Clostridium cochlearium</i> , <i>C. welchii</i> ; <i>Enterococcus faecalis</i> ; <i>Staphylococcus aureus</i> ; <i>Streptococcus epidermidis</i> , <i>S. pyrogenes</i>	993
Spiders	Gram-negative bacteria	<i>Acinetobacter calcoaceticus</i> ; <i>Citrobacter freundii</i> ; <i>Enterobacter aerogenes</i> ; <i>Morganella morganii</i>	984
	Gram-positive bacteria	<i>Staphylococcus</i> spp. (coagulase-negative)	984
Mites, midges	Gram-negative bacteria	<i>Acinetobacter calcoaceticus</i> ; <i>Burkholderia cepacia</i> ; <i>Enterobacter agglomerans</i> , <i>E. aerogenes</i> ; <i>Hafnia alvei</i> ; <i>Pseudomonas aeruginosa</i>	984
	Gram-positive bacteria	<i>Staphylococcus</i> spp. (coagulase-negative)	984
Mosquitoes	Gram-negative bacteria	<i>Acinetobacter calcoaceticus</i> ; <i>Enterobacter cloacae</i>	984
	Gram-positive bacteria	<i>Enterococcus</i> spp.; <i>Staphylococcus</i> spp. (coagulase-negative)	984

Appendix E - Evaluation Plan Elements

Process measurement is expected to be the focus of efforts to monitor compliance with the evidence-based Category IA or IB recommendations provided in Part II of this guideline. The following elements can be used for this purpose.

Table E.1. Process Measurement Elements^a

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- ☒ Conduct risk assessment prior to construction, renovation, demolition, or major repair projects.
 - ☒ Conduct ventilation assessments related to construction barrier installation.
 - ☒ Establish and maintain appropriate pressure differentials for special care areas (e.g., operating rooms, airborne infection isolation, protective environments).
 - ☒ Evaluate non-tuberculous mycobacteria culture results for possible environmental sources.
 - ☒ Implement infection control procedures to prevent environmental spread of antibiotic-resistant gram-positive cocci and assure compliance with these procedures.
-

a. Based on Category IA or IB recommendations

Appendix F - Information Resources

The following sources of information may be helpful to the reader. Some of these are available at no charge, while others are available for purchase from the publisher.

AIR and WATER

1. Jensen PA, Schafer MP. Sampling and characterization of bioaerosols. NIOSH Manual of Analytical Methods; revised 6/99. <http://www.cdc.gov/niosh/nmam/pdfs/chapter-j.pdf>
2. American Institutes of Architects. *Guidelines for Design and Construction of Hospital and Health Care Facilities*. Washington DC; American Institute of Architects Press; 1996.
(Note: a new edition of this document is due to be available in the spring of 2001.)
AIA, 1735 New York Avenue, NW, Washington DC 20006.
3. ASHRAE. Standard 62, and Standard 12-2000. These documents may be purchased from: American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc. 1791 Tullie Circle, NE, Atlanta GA 30329 1-800-527-4723 or (404) 636-8400
4. University of Minnesota websites: <http://www.dehs.umn.edu>
Indoor air quality site: <http://www.dehs.umn.edu/resources.htm#indoor>
Water infiltration and use of the wet test meter: <http://www.dehs.umn.edu/remangi.html>

ENVIRONMENTAL SAMPLING

1. ISO. Sterilization of medical devices - Microbiological methods, Part 1. ISO standard 11737-1. Paramus NJ; International Organization for Standardization; 1995.

REGULATED MEDICAL WASTE

1. U.S. Environmental Protection Agency. This is the address for their link page on their Internet web site which can direct you to any state for information about medical waste rules and regulations at the state level: <http://www.epa.gov/epaoswer/other/medical/stregs.htm>

GENERAL RESOURCES

In addition to several excellent infection control and epidemiology textbooks:

1. APIC Text of Infection Control and Epidemiology. Association for Professionals in Infection Control and Epidemiology, Inc. Washington DC; 2000. (Two binder volumes, or CD-ROM)
2. Abrutyn E, Goldmann DA, Scheckler WE. Saunders Infection Control Reference Service, 2nd Edition. Philadelphia PA; WB Saunders; 2000.

Appendix G - Areas of Future Research and Guideline Development

AIR

Research

1. Standardize the methodology and interpretation of microbiologic air sampling.
2. Determine “action levels” or minimum infectious dose for aspergillosis.
3. Develop better molecular typing methods to better define the epidemiology of healthcare-associated outbreaks of aspergillosis, to associate clinical and environmental isolates.
4. Determine the incubation period of aspergillosis.
5. Develop new methods of diagnosis aspergillosis that can reliably lead to early recognition of infection.
6. Continue to define the risk factors for aspergillosis among all categories of patients.
7. Evaluation of the pathogenicity of other opportunistic fungi such as *Fusarium*, *Alternaria*, and other emerging opportunistic fungi for airborne transmission potential.
8. Develop more specific, less toxic antifungal therapies for invasive fungal infections.
9. Establish correlation between recovery of *Bordetella pertussis*, respiratory syncytial virus, *Pneumocystis carinii* and other microorganisms by polymerase chain reaction (PCR) as proof of airborne transmission with clinical significance.
10. Assess the value of laminar flow for surgeries other than orthopedic surgery.
11. Determine the significance of bacteria and fungi isolated by particulate sampling around the surgical field and their contribution to postoperative surgical site infections.
12. Determine if particulate sampling can be routinely performed in lieu of microbiologic sampling for purposes such as determining air quality of clean environments (i.e., operating rooms, HSCT units).
13. Generate epidemiologic data to support recommendations for specific values of air changes per hour (6 ACH vs. 12 ACH)
14. Determine if there is any decrease to the time required for airborne contaminant removal when portable HEPA filter units are placed in a room of defined ACHs.

Guideline Development

1. Formulate guidelines for care of patients in need of surgical procedures while confined to airborne isolation or protective environments.
2. Develop recommendations for preventing transmission of environmental airborne fungi for immunocompromised patients.

WATER

Research

1. Continue studies to evaluate new methods of water treatment, both in the facility and at the water utility (e.g., ozone, chlorine dioxide, copper/silver, monochloramines).
2. Continue to evaluate the role of biofilms in overall water quality and determine the epidemiology of biofilms and their contribution to transmission of waterborne disease.
3. Continue to examine the hot water temperature issue to help prevent future outbreaks of legionellosis.
4. Continue to develop strategies for preventing outbreaks of waterborne protozoan disease (i.e., cryptosporidiosis, giardiasis).
5. Continue to define the epidemiology of healthcare-associated legionellosis, including the risk factors for all categories of patients.
6. Continue to evaluate methods for reprocessing dialyzers.
7. Determine if the use of ultrapure fluids in dialysis is feasible and warranted (i.e., need to determine action levels for the final bath).
8. Conduct studies to determine the relationship between environmental isolation of organisms (e.g., *Legionella* spp.) and the rate of disease.
9. Conduct studies to determine the most cost-effective means of preventing healthcare-associated legionellosis.

Guideline Development

1. Encourage AAMI to finalize their guidance on hemodialysis water quality.
2. Continue to evaluate the impact of water sampling for *Legionella* spp., especially in high-risk environments, on the prevention of transmission of legionellosis.

ENVIRONMENTAL SERVICES

Research

1. Continue to evaluate the innate resistance of microorganisms to the action of germicidal chemicals.
2. Determine what, if any, linkage there is between antibiotic resistance and resistance to disinfectants.
3. Continue to define the contributions of surfaces and hand transfer in the epidemiology of healthcare-associated bacterial infections.
4. Conduct research into prion inactivation using more modern sterilization methodologies.

LAUNDRY AND BEDDING

Research

1. Continue to evaluate microbial contamination of fabrics and determine the contribution of such fabrics in the epidemiology of healthcare-associated diseases.
2. Evaluate the microbial inactivation capabilities of the newer laundry detergents, bleach substitutes, and other laundry additives.

ANIMALS IN HEALTHCARE FACILITIES

Research

1. Conduct surveillance to monitor incidence of infections among patients whose facilities use animal programs and conduct investigations to determine new infection control strategies.
2. Evaluate the impact of permitting procedures performed on animals (i.e., surgery, imaging) that occur in human healthcare facilities.

REGULATED MEDICAL WASTE

Research

1. Explore new ways to treat medical waste, to minimize its volume.
2. Explore ways to re-enable healthcare facilities to treat cultures and stocks on-site.