Airborne Respiratory Diseases and Mechanical Systems for CONTROL OF MICROBES

Airborne respiratory pathogens and diseases in health care facilities are numerous and dangerous. HVAC systems are critical in controlling them.

By W. J. Kowalski, PE, Graduate Researcher, and ***William Bahnfleth, PhD, PE,*** *Assistant Professor, The Pennsylvania State University, Architectural Engineering Dept., University Park, Pa.*

Airborne transmission of respiratory diseases in indoor environments remains a problem of indoor air quality (IAQ) with few engineering alternatives and for which performance goals and design parameters are unclear. The engineer who attempts to deal with microbial IAQ finds that pertinent microbiological information exists in abundance but not in easily digestible forms. This article summarizes the relevant literature of medical microbiology and aerobiology in a manner that engineers may find useful and informative and that will facilitate the design of HVAC systems intended to reduce the threat. The general principles presented here can be applied to any indoor environment, including office buildings, schools, residences, hospitals, and isolation wards.

Origin of respiratory diseases

The first indoor environments, built by man over half a million years ago, included caves with leather-draped interiors, fur-carpeted tents, and huts covered with animal hides. Microbial predators existed from time immemorial, but transmission had always required direct contact because they could not tolerate the sunlight and temperature extremes outdoors. Man's cozy new habitats made it possible for these ancient parasites to survive short airborne trips between hosts.

Animal husbandry seems to have resulted in a number of pathogens jumping species and then becoming adapted to indoor transmission to the exclusion of outdoor transmission. These include rhinoviruses, diptheria, TB, smallpox, measles, and influenza, which appear to have come variously from horses, cows, dogs, pigs, and chickens. Most contagious human pathogens have evolved to such dependence on man's habitats for transmission that they lack any ability to survive outdoors for long.[1]

In contrast, the non-contagious pathogens, including the fungi, environmental bacteria, and some animal pathogens, have maintained the ability to survive in the environment. Even so, direct sunlight is rapidly fatal to almost anything but spores.[1]

Classification of pathogens

Pathogens are any disease-causing microorganism, but the term applies to any microbial agent of respiratory irritation, including allergens or toxigenic fungi. Respiratory pathogens fall into three major taxonomic groups: viruses, bacteria, and fungi. The fungi and some bacteria, most notably the actinomycetes, form spores. Since spores are characteristically larger and more resistant to factors that will destroy viruses and bacteria, the engineer may find it more convenient to consider spores a definitive and separate category.

The single most important physical characteristic by which to classify airborne pathogens is size since it directly impacts filtration efficiency.[2] Fig. 1 presents a graphic comparison of airborne respiratory pathogens in which the spores, bacteria, and viruses can be observed to differentiate well, based on size alone. The left axis indicates the "average" or typical diameter or width. The areas of the circles do not represent the actual sizes of the microbes, but each represents the diameter in proportion to one another. The span of diameters is seen to be almost four orders of magnitude. Some microbes are oval or rod-shaped, and for these only, the smaller dimension is indicated.

[1]*Superscript numerals indicate references listed at end of article.*

Perhaps the most important classification is that of communicable versus non-communicable, a distinction that has both medical and engineering relevance. The term *communicable* is synonymous with the term *contagious*. Communicable diseases come mainly from humans, while non-communicable diseases hail mostly from the environment. However, many microbes that are endogenous to humans or are environmentally common may cause opportunistic infections in those whose health has been compromised. These occur primarily as nosocomial, or hospital-acquired, infections. These three categories then define all airborne pathogens:

- Communicable
- Non-communicable
- Primarily nosocomial

Table 1 lists all respiratory pathogens under these three categories, along with major diseases, common sources, and average diameters. In the column identifying microbial group, the term *actinomycetes* refers only to the spore-forming actinomycetes. Some general observations can be made from these charts such as the fact that most contagious pathogens come from humans, most non-contagious pathogens come from the environment, and most primarily nosocomial infections tend to be endogenous. These tables are not necessarily inclusive since a number of pathogens, such as *E. coli*, *Bacillus subtilis*, and some other strains of *Legionella*, can, on rare occasions, cause respiratory disease or allergic reactions.[3] The abbreviation "spp." denotes that infections may be caused by more than one species of the genera but does not imply that all species are pathogenic.

Table 1 lists only respiratory pathogens, although non-respiratory pathogens can also be airborne. Certain infections of the skin or eyes, nosocomial infections of open wounds and burns, and contamination of medical equipment may occur by the airborne route. Although these types of infections have not been well studied, any pathogen that transmits by the airborne route will be subject to the same principles and removal processes described in this article.

Communicable diseases

Table 1 lists all the main respiratory diseases that can transmit between human hosts via the airborne route. Humans are the natural reservoir for most contagious pathogens but some notable exceptions exist. Pneumonic plague and Arenavirus epidemics originate with rodents or other mammals.[1] In regards to the mysterious origin of *Influenza*, humans apparently share the function of natural reservoir with birds and pigs, as strains of this virus periodically jump between species.[3]

continued on page 37

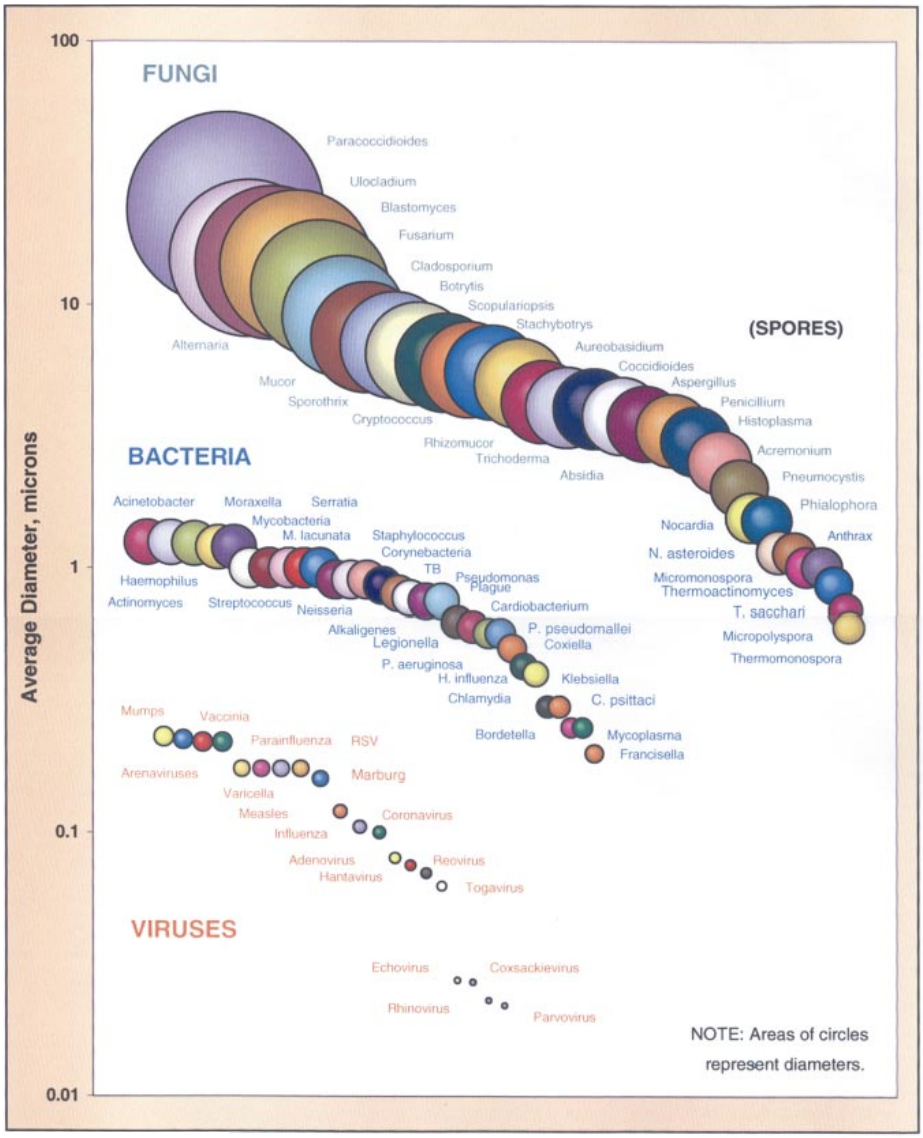

Figure 1. Relative size of airborne respiratory pathogens.

Table 1: Communicable Respiratory Pathogens

AIRBORNE PATHOGEN	MICROBIAL GROUP	DISEASE	SOURCE	Diameter microns	Notes
Adenovirus	VIRUS	colds	Humans	0.08	
Arenavirus	VIRUS	hemorrhagic fever	Rodents	0.18	F
Coronavirus	VIRUS	colds	Humans	0.11	
Coxsackievirus	VIRUS	colds	Humans	0.027	
Echovirus	VIRUS	colds	Humans	0.028	
Morbillivirus	VIRUS	measles (rubeola)	Humans	0.12	F, N
Influenza	VIRUS	flu	Humans, birds	0.1	F, N
Parainfluenza	VIRUS	flu	Humans	0.22	N
Paramyxovirus	VIRUS	mumps	Humans	0.23	F, N
Parvovirus B19	VIRUS	fifth disease, anemia	Humans	0.022	F
Reovirus	VIRUS	colds	Humans	0.075	
Respiratory Syncytial Virus	VIRUS	pneumonia	Humans	0.22	F, N
Rhinovirus	VIRUS	colds	Humans	0.023	
Togavirus	VIRUS	rubella (German measles)	Humans	0.063	N
Varicella-zoster	VIRUS	chickenpox	Humans	0.16	N
Chlamydia pneumoniae	BACTERIA	pneumonia, bronchitis	Humans	0.3	N
Mycobacterium tuberculosis	BACTERIA	TB	Humans	0.86	F, N
Yersinia pestis	BACTERIA	pneumonic plague	Rodents	0.75	F

Table 1: Primarily Nosocomial Respiratory Pathogens

AIRBORNE PATHOGEN	MICROBIAL GROUP	DISEASE	SOURCE	Diameter microns	NOTES
Acinetobacter	BACTERIA	opportunistic infections	Environmental	1.3	E, N
Actinomyces israelii	BACTERIA	actinomycosis	Humans	1.0	E, N
Alkaligenes	BACTERIA	opportunistic infections	Humans	0.75	E, N
Bordetella pertussis	BACTERIA	whooping cough	Humans	0.25	E, N
Cardiobacterium	BACTERIA	opportunistic infections	Humans	0.63	E, N
Corynebacteria diphtheria	BACTERIA	diptheria	Humans	1.0	E, N
Haemophilus influenzae	BACTERIA	meningitis, pneumonia	Humans	0.43	E, N, F
Haemophilus parainfluenzae	BACTERIA	opportunistic infections	Humans	1	E, N
Klebsiella pneumoniae	BACTERIA	opportunistic infections	Environmental	0.4	E, N
Moraxella catarrhalis	BACTERIA	opportunistic infections	Humans	1.3	E, N
Moraxella lacunata	BACTERIA	opportunistic infections	Humans	1	E, N
Mycobacterium avium	BACTERIA	cavitary pulmonary dis.	Environmental	1.2	N
Mycoplasma pneumoniae	BACTERIA	pneumonia	Humans	0.25	E, N
Neisseria meningitidis	BACTERIA	meningitis	Humans	0.8	E, N, F
Pseudomonas aeruginosa	BACTERIA	opportunistic infections	Environmental	0.57	N
Pseudomonas mallei	BACTERIA	opportunistic infections	Environmental	0.77	N
Pseudomonas pseudomallei	BACTERIA	opportunistic infections	Environmental	0.57	N
Serratia marcescens	BACTERIA	opportunistic infections	Environmental	1.3	E, N
Staphylococcus aureus	BACTERIA	opportunistic infections	Humans	1	E, N
Streptococcus pneumoniae	BACTERIA	pneumonia, otitis media	Humans	0.9	E, N, F
Streptococcus pyogenes	BACTERIA	scarlet fever, pharyngitis	Humans	0.9	N
Pneumocystis carinii	Protozoa / Fungi	pneumocystosis	Environmental	2	S, N
Cryptococcus neoformans	FUNGI	cryptococcosis	Environmental	5.5	S, N

NOTES:
E = Endogenous, common as human flora
F = Fatalities occur (excluding nosocomial)
HP = Hypersensitivity Pneumonitis
N = Nosocomial, common as (purple blocks)
EAA = Extrinsic Allergic Alveolitis
S = Spores
VOC = Volatile Organic Compounds produced
References: 4, 8, 17, 18, 22

Table 1: Non-Communicable Respiratory Pathogens

AIRBORNE PATHOGEN	MICROBIAL GROUP	DISEASE common (potential)	SOURCE	Diameter microns	NOTES
Hantavirus	VIRUS	hantavirus	Rodents	0.07	F
Poxvirus - Vaccinia	VIRUS	cowpox	Agricultural	0.23	
Bacillus anthracis	BACTERIA	anthrax	Cattle, sheep	1.1	S, F
Chlamydia psittaci	BACTERIA	psittacosis	Birds	0.3	
Coxiella burnetii	BACTERIA	Q fever	Cattle, sheep	0.5	
Francisella tularensis	BACTERIA	tularemia	wild animals	0.2	F
Legionella pneumophila	BACTERIA	LD, Pontiac fever	Environmental	0.6	F, N
Mycobacterium intracellulare	BACTERIA	cavitary pulmonary dis.	Environmental	1.2	
Mycobacterium kansasii	BACTERIA	cavitary pulmonary dis.	unknown	0.86	
Micromonospora faeni	ACTINOMYCETES	Farmer's Lung, HP	Agricultural	1	S
Micropolyspora faeni	ACTINOMYCETES	Farmer's Lung, HP	Agricultural	0.69	S
Nocardia asteroides	ACTINOMYCETES	nocardiosis	Environmental	1.1	S, N
Nocardia brasiliensis	ACTINOMYCETES	pulmonary mycetoma	Environmental	1.5	S, N
Nocardia caviae	ACTINOMYCETES	nocardiosis	Environmental	1.5	S, N
Thermoactinomyces sacchari	ACTINOMYCETES	bagassosis, HP	Agricultural	0.86	S
Thermoactinomyces vulgaris	ACTINOMYCETES	Farmer's Lung, HP	Agricultural	1	S
Thermomonospora viridis	ACTINOMYCETES	Farmer's Lung, HP	Agricultural	0.6	S
Absidia corymbifera	FUNGI	zygomycosis	Environmental	3.8	S
Acremonium spp.	FUNGI	(EAA)	Environmental	2.5	S
Alternaria alternata	FUNGI	mycotoxicosis	Environmental	14.4	S
Aspergillus spp.	FUNGI	aspergillosis, VOC	Environmental	3.5	S, N
Aureobasidium pullulans	FUNGI	chromomycosis, EAA	Environmental	5	S
Blastomyces dermatitidis	FUNGI	blastomycosis	Environmental	14	S, N
Botrytis cinera	FUNGI	EAA	Environmental	7	S
Chaetomium globosum	FUNGI	chromomycosis, VOC	Environmental	5.5	S
Cladosporium spp.	FUNGI	chromoblastomycosis	Environmental	9	S
Coccidioides immitis	FUNGI	coccidioidomycosis	Environmental	4	S, N
Emericella nidulans	FUNGI	(mycotoxicosis)	Environmental	3.3	S
Epicoccum nigrum	FUNGI	(EAA)	Environmental	20	S
Eurotium spp.	FUNGI	EAA	Environmental	5.8	S
Exophiala jeanselmei	FUNGI	chromomycosis	Environmental	2.0	S
Fusarium spp.	FUNGI	mycotoxicosis, VOC	Environmental	11.5	S
Geomyces pannorum	FUNGI	EAA	Environmental	3	S
Helminthosporium	FUNGI	EAA	Environmental	12.5	S
Histoplasma capsulatum	FUNGI	histoplasmosis	Environmental	3	S, N
Mucor plumbeus	FUNGI	mucormycosis	Environmental	7.5	S, N
Paecilomyces variotii	FUNGI	mycotoxicosis	Environmental	3	S
Paracoccidioides brasiliensis	FUNGI	paracoccidioidomycosis	Environmental	23	S
Penicillium spp.	FUNGI	mycotoxicosis, VOC	Environmental	3.3	S
Phialophora spp.	FUNGI	chromomycosis	Environmental	1.5	S
Phoma spp.	FUNGI	mycotoxicosis	Environmental	3.3	S
Rhizomucor pusillus	FUNGI	zygomycosis	Environmental	4.3	S
Rhizopus stolonifer	FUNGI	zygomycosis	Environmental	8	S, N
Rhodoturula spp.	FUNGI	(EAA)	Environmental	14	S
Scopulariopsis spp.	FUNGI	onychomycosis	Environmental	6	S
Sporothrix schenckii	FUNGI	sporotrichosis	Environmental	6.5	S
Stachybotrys spp.	FUNGI	stachybotryotoxicosis	Environmental	5.7	S, F
Trichoderma spp.	FUNGI	mycotoxicosis, VOC	Environmental	4.1	S
Ulocladium spp.	FUNGI	EAA	Environmental	15	S
Wallemia sebi	FUNGI	EAA	Environmental	3	S

continued from page 35

Many contagious respiratory pathogens also transmit by direct contact through the exchange of infectious droplets or particles called fomites.[4] The eyes and nasal passages are vulnerable to fomite transmission. The predominance of these direct routes in comparison with the inhalation route has not been well established but can be very species-dependent.[5] Infectivity is also lost upon drying, and therefore hand or surface contact may require the exchange of moisture as well as an infectious dose.[1,6]

Barely 20 pathogens account for the overwhelming number of contagious respiratory infections. Table 2 lists the characteristics of these infections, while the typical course of these infections is depicted in Fig. 2. The infection rate refers to the fraction of those exposed to an infectious dose who contract the disease. This type of information can be useful to engineers attempting risk assessment or procedural control of infectious occupants or patients. Few infectious doses have been established, but for purposes of making rough or conservative estimations, as few as 1-10 TB bacilli can be infectious for humans—while a total of 200 Rhinovirus virions may be required to cause a cold.[4]

Most respiratory parasites induce their hosts to aerosolize large quantities of infectious bioaerosols by nasopharyngeal irritation, which causes coughing and sneezing.[4,5] Consider the profiles of the particle sizes shown in Fig. 3. A single sneeze can generate a hundred thousand floating bioaerosol particles, and many may contain viable microorganisms.[7] A single cough typically produces about one percent of this amount, but coughs occur about 10 times more frequently than sneezes.[7] Bioaerosols produced by talking are negligible, but extended shouting and singing can transmit infections.

Some limited data from Duguid[7] is available in generation rates stating that A TB infective can produce 1-249 bacilli per hr,[8] while a person in the infectious stage of a cold may produce 6200 droplet nuclei per hr containing viable viruses that remain airborne longer than 10 min. In one measles epidemic, 5480 virions were generated per hr.[8]

The dose received from an airborne concentration of microbes could be considered a factor under engineering control since it

TABLE 2: Communicable Respiratory Infection Characteristics

PATHOGEN		Common Disease	Annual Cases (U.S.)	Average Incubation (days)	Infectious Peak (days)	Maximum Duration (days)	Infection Rate (fraction)
VIRUS	BACTERIA						
Adenovirus		colds	*	4	4-9	19	0.51-0.75
Coronavirus		colds	*	3	(3-4)	18	0.45-0.5
Coxsackievirus		colds	*	3	3-12	20	0.53-0.64
Echovirus		colds	*	3	(3-4)	18	0.43-0.80
Influenza		flu	200000*	2	2-7	21	0.2-0.8
Morbillivirus		measles	25000	12	8-16	29	0.85
Parainfluenza		flu	*	3	3-12	21	0.2-0.75
Paramyxovirus		mumps	10000	17	10-26	39	0.6-0.85
Parvovirus B19		fifth disease	-	8	7-14	28	0.3-0.6
Respiratory Synctial Virus		pneumonia	*	2	5-7	14	0.5-0.9
Rhinovirus		colds	*	2	2-7	7	0.38-0.89
Togavirus		rubella	3000	17	24-31	31	0.3-0.8
Varicella-zoster		varicella	*	16	12-20	25	0.75-0.96
Bordetella pertussis		whpg cough	2000	8	15-22	42	high
Chlamydia pneumoniae		pharyngitis	-	7	7-21	28	0.5
Corynebacteria diptheria		diptheria	490000	3	2-10	10	varies
Haemophilus influenzae		meningitis	8000	3	3-4	(14)	0.2-0.5
Mycobacterium tuberculosis		TB	21000	28	varies	--	0.33
Neisseria meningitidis		meningitis	4500	3	3-4	(21)	high
Streptococcus pneumoniae		pneumonia	500000	2	(2-10)	21	0.1-0.3
Yersinia pestis		pn. plague	14	2	2-3	3	varies

References 4. 8. 10. 17. 22

*(Common respiratory infections are often not reported.)

depends on the local air change rate and degree of mixing as well as the generation rate. The successful transmission of an infection, however, depends on all of the following factors:

- Susceptibility of the individual (immunity).
- Duration of exposure.
- Concentration of infectious agent.
- Virulence of infectious agent.
- Breathing rate.
- Route of infection (inhalation, eyes, nasopharynx, etc.).

None of these factors is necessarily an absolute determinant. Health and degree of immunity can be as important as the dose received from prolonged exposure.

Computations of infectious airborne doses can be fraught with uncertainty. Epidemiological studies on colds avoid these problems by computing actual risks. Fig. 4 shows how duration and proximity to an infectious person can increase the likelihood of infection, based on data from Lidwell's studies of the common cold.[9] These data suggest that there may be a threshold distance beyond which risk decreases sharply. This risk may result from local airborne concentrations but may also include the risk of contact with fomites.

Non-communicable diseases

The list of non-communicable pathogens in Table 1 includes all known that cause respiratory infections, allergic reactions, and toxic reactions. Included among the diseases are EAA and HP (see notes),

continued on page 40

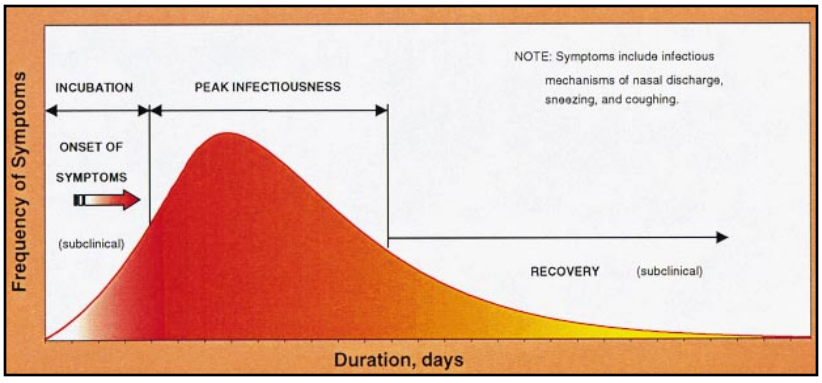

Figure 2. Generic curve for duration of symptoms of respiratory infections

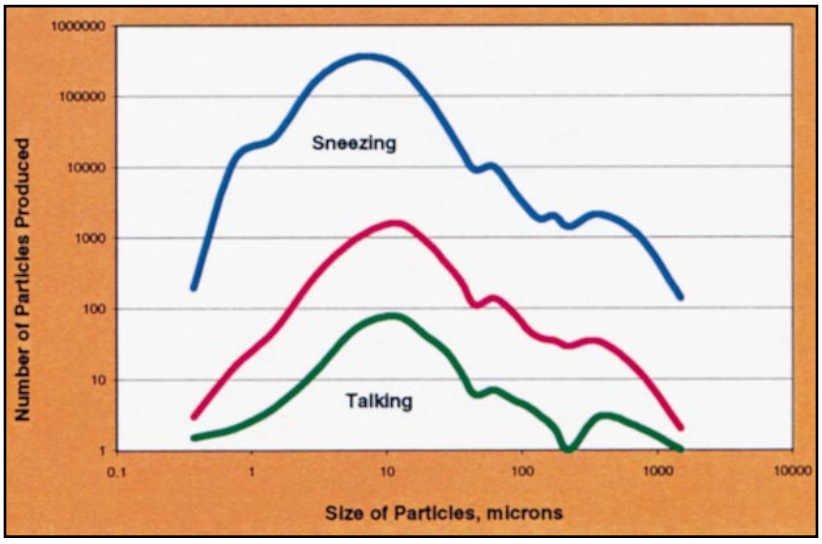

Figure 3. Profile of particle sizes produced by an infectious person.
Based on data from Duguid et al 1945.

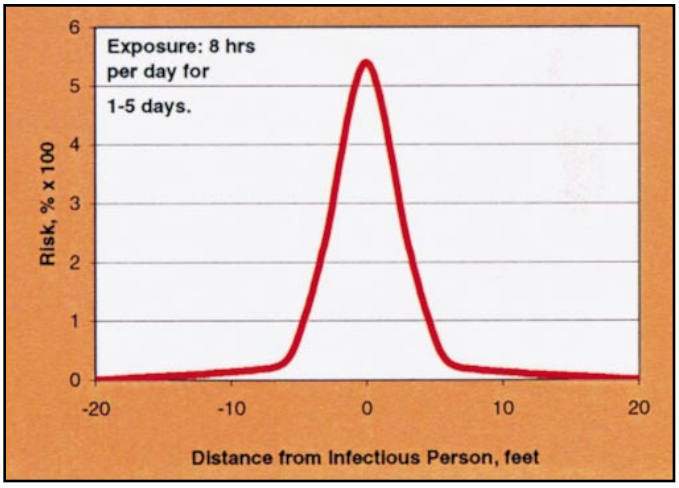

Figure 4. Risk of cold infection from proximity. Risk at zero represents intimate (husband-wife) contact. *Estimated per data from Lidwell.*[9]

continued from page 38

which are sometimes associated with sick building syndrome (SBS). Non-communicable infections are almost entirely due to fungal or actinomycete spores and environmental or agricultural bacteria.

Spores form the most important group of non-communicable diseases. Outdoor spore levels vary with season and climate and can reach very high levels when dry, windy conditions result in disturbance of the soil where fungi grow. Surprisingly, few cases of respiratory infection have ever been attributed to inhalation of outdoor air,[4,5] probably because most people, especially Americans and Europeans, spend over 90 percent of their time indoors.[10] A small proportion of actinomycete infections have occurred outdoors in agricultural facilities, although most tend to occur inside barns and worksheds.[11,12]

Indoor air spore levels can differ from outdoor air in both concentration and composition of spores. In normal, dry buildings, spore levels tend to be anywhere from 10 to 100 percent of outdoor spore levels[11] and are mostly less than 200 colony forming units (CFU) per cu meter. Problem-free, multi-story office buildings typically have levels that are 10 to 31 percent of the outdoor air[11] levels. The composition of fungal species indoors tends to reflect that of the outdoors.[13] Some fungal species, most notably A*spergillus* and *Penicillium*, are often found to account for 80 percent of indoor spores.[10]

Spores will germinate and grow in the presence of moisture and nutrients[13] in locations such as basements, drain pans, and on refrigerator coils. As a result of such growth, spores can be generated internally in problem buildings, wet buildings, and certain agricultural facilities at a high enough rate to cause indoor spore levels to exceed outdoor levels. If spore concentrations indoors consistently exceed

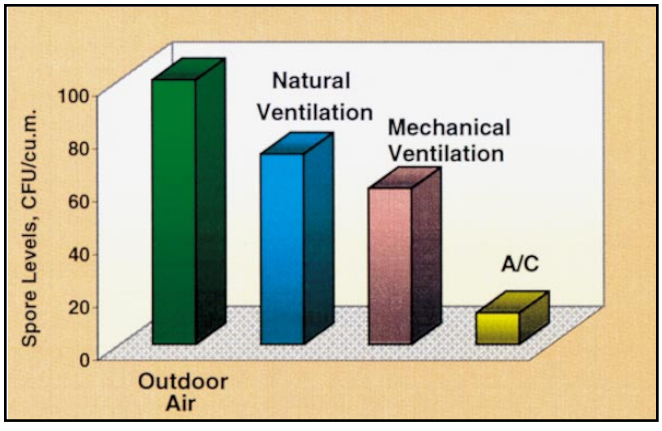

Figure 5. Indoor spore levels by ventilation system type. *From the California Healthy Buildings Study.*[11]

Table 3: Microbial Levels in Indoor and Outdoor Air

(Suggested Guidelines, Upper Limits, Average or Representative Levels)

Microbe	Lower Limit	Average Range		Upper	Qualification	I/O Ratio		Reference
	CFU/cu.m.	Low	High	Limit		Low	High	
Indoor Fungal Spores*	50	10	500	150		0.1	0.33	20, 23(ACGIH), 25(CG)
(summer)				500				25(CG)
Outdoor Fungal Spores	100	100	1000	-		-	-	9(CHBS), 25
Actinomycete Spores	0	0	150	240	normal homes	-	-	23(Nevalainen), 25
Outdoor Actino. Spores	0	4	-	-	farmhouses	-	-	23 (Heineman)
Bacteria, non-pathogenic	50	0	500	50		0.26	1.1	10, 23, 25(ACGIH)
Outdoor Bacteria	-	179	1083	-		-	-	9(Brickus), 23
Pathogenic Bacteria	0	-	-	0				20(AIHA)
Viruses	0	-	-	0				20(AIHA)

*(when species mix reflects outdoor air)
CG: Canadian Guidelines

ACGIH: American Conference of Government Industrial Hygienists
AIHA: American Industrial Hygiene Association
CHBS: California Healthy Buildings Study

outdoor levels, the building can be inferred to contain an indoor amplifier.[14]

In the California Healthy Buildings Study,[11] naturally ventilated, mechanically ventilated, and air conditioned buildings all had lower indoor spore levels than the outdoors (Fig. 5). However, Fig. 5 may reflect favorable local conditions since many studies have measured much higher levels than these in non-problem buildings.

Table 3 lists the results of various studies that include measurements of outdoor spore levels and typical, average, or representative indoor levels. These levels do not necessarily pose a health threat. Measurements and guidelines vary almost as widely as outdoor levels vary seasonally and geographically.

Microorganisms will take advantage of any opportunity to establish themselves and multiply in a new environment.[4] Niches for microbial growth may be created inadvertently by engineered systems that generate moisture such as humidifiers, evaporative air coolers, cooling coil drain pans, and condensation on ductwork insulation. Amplification may result in airborne concentrations above the outdoors[10] and may reach unhealthy levels.[13] Legionnaire's Disease provides a sentinel example of pathogenic microbial amplification by an engineered system.

Amplifying factors can be controlled through various means, including preventive design through humidity and moisture control. Some other first and second line defensive measures include filtration, the removal of materials that provide nutrients, procedural cleaning and maintenance, and the use of biocidal equipment.

Table 4 identifies fungal pathogens that have been found to grow indoors on various surfaces or in HVAC equipment. Unidentified multiple species (spp.) may not necessarily be pathogenic. Many factors may dictate which pathogens will grow indoors such as climate, indoor materials, degree of human occupancy, hygiene, and moisture levels.[1,8]

Table 5 identifies some pathogenic environmental bacteria that have been found growing indoors or on HVAC equipment. Occasionally, some contagious bacteria disemminated from hu-

Table 4: Fungi That May Grow Indoors

Airborne Pathogen	Indoor Growth		HVAC Equipment Growth	
	Location	Reference	Location	Reference
Acremonium spp.			humidifier water	Heineman (23)
			HVAC fiberglass insulation	(19)
Alternaria spp.	indoors > outdoors	(2)	cooling systems	(11)
	paint mildew	(26)	refrigerator coils	(21)
	carpet dust	Gravesen (23)	filters	Shata (9), (12)
	floor dust	Hoekstra (23)	filters	Neumeister (16)
			dust in ductwork	Valbjorn (9)
Aspergillus spp.	indoors > outdoors	(2)	evaporative air cooler	(16)
	carpet dust	Gravesen (23)	HVAC fiberglass insulation	(19)
	floor dust	Hoekstra (23)	cooling systems, coils	(11), (30)
			fans, filters	Heineman (23) , (12)
			dust in ductwork	Sugawara (16)
Aureobasidium pullulans	moist building materials	Pasanen (23)	filters	Shata (9)
	latex painted surfaces	(26)		
Chaetomium spp.			HVAC fiberglass insulation	(19)
			filters	Heineman (23)
			dust in ductwork	Valbjorn (9)
Cladosporium spp.	wet carpet, wet walls	(21)	evaporative coolers	(11)
	moist building materials	Pasanen (23)	HVAC fiberglass insulation	(19)
	latex painted surfaces	(26)	filters	Shata (9)
	floor dust	Hoekstra (23)	HVAC metal surfaces	(1)
	carpet dust	Gravesen (23)	fans, filters	Heineman (23)
			ductwork dust	Sugawara (16)
Cryptococcus spp.	floor dust	Hoekstra (23)		
Epicoccum spp.	indoors > outdoors	Kemp (16)	fiberglass insulation	Morey (9)
Eurotium herbariorum	gypsum-based finishes	Adan (23)		
Exophiala spp.			humidifier water	Heineman (23)
Fusarium spp.	indoors > outdoors	Fouad (16)	filters	Neumeister (16)
	floor dust	Hoekstra (23)	humidifier water	Heineman (23)
Helminthosporium	indoors > outdoors	Kemp (16)		
Mucor spp.	indoors > outdoors	Kemp (16)	fans, filters	Heineman (23) , (12)
	floor dust	Hoekstra (23)	dust in ductwork	Valbjorn (9)
Paecilomyces spp.			humidifier water	Heineman (23)
Penicillium spp.	indoors > outdoors	(2)	air conditioners	(11)
	latex painted surfaces	(26)	evaporative air cooler	(16)
	carpet dust	Gravesen (23)	HVAC ducts	(5)
			filters	Pasanen (23), (12)
			fans, humidifier water	Heineman (23)
Phialophora spp.			humidifier water	Heineman (23)
Phoma spp.	paint mildew	(26)	filters	Neumeister (16)
	floor dust	Hoekstra (23)	humidifier water	Heineman (23)
Rhizopus spp.	floor dust	Hoekstra (23)	fans	Heineman (23)
			filters	Neumeister (16)
			dust in ductwork	Valbjorn (9)
Rhodoturula spp.	wet carpet, wet walls	(21)		
	indoors > outdoors	Kemp (16)		
Scopulariopsis spp.	floor dust	Hoekstra (23)	filters	Heineman (23)
Stachybotris spp.	building materials	Scott (16)	fans, humidifier water	Heineman (23)
	moist building materials	Pasanen (23)		
Trichoderma spp.	indoors	(7)	fans	Heineman (23)
	moist building materials	Pasanen (23)	filters	Neumeister (16)
			ductwork dust	Sugawara (16)
Ulocladium spp.	floor dust	Hoekstra (23)	filters	Neumeister (16)
			humidifier water	Heineman (23)
Wallemia sebi	floor dust	Hoekstra (23)	filters	Shata (9)

mans can be found in water, equipment, or in dust, but these are transient occupants and unlikely to grow or survive long outside of human hosts.[1,5]

Nosocomial infections

All respiratory pathogens are potentially nosocomial, but those that occur almost exclusively as nonsocomial infections are listed in Table 1 such as primarily nosocomial respiratory pathogens.

Table 5: Bacteria That May Grow Indoors

Airborne Pathogen	Location of Growth	Reference
Acinetobacter	potable water	Highsmith (14)
Klebsiella pneumoniae	potable water	Highsmith (14)
Legionella pneumophila	potable water	(22)
	cooling towers	(14)
Micropolyspora faeni	home humidifiers	(7)
Pseudomonas aeruginosa	indoors	Strom (9)
	indoor dust	(18)
	potable water	Highsmith (14)
	evaporative air cooler	(16)
	humidifiers	(7)
Pseudomonas spp.	filters	Martikainen (9)
Serratia Marcescens	potable water	Highsmith (22)
Thermoactinomycetes vulgaris	air conditioners	(7)
	humidifier water	Heineman (23)

The other common nosocomial infections are identified with a purple boxed *N* in the notes column.

In intensive-care units, almost a third of nosocomial infections are respiratory, but not all of these are airborne since some are transmitted by contact or by intrusive medical equipment.[15] Nosocomial infections can also be airborne but non-respiratory such as when common microbes like *Staphylococcus* settle on open wounds, burns, or medical equipment.

Patients who succumb to nosocomial infections are often those whose natural defenses have been compromised either as a result of disease, medication, injury, or bypassed by intrusive procedures. In cases of immune system deficiency, even a patient's own endogenous flora could cause infection, while normally benign environmental microbes can become pathogenic.

The protection of patients from potential pathogens requires the reduction of microbial contaminants below normal or ambient levels. This is usually accomplished through the use of isolation rooms, HEPA filters, UVGI, and strict hygiene procedures.[15] In the health care environment, particular attention must be paid to the possibility of microbial growth indoors and in the air handling units, even if levels are not a threat to healthy people. Low-level indoor microbial amplification in health care settings may cause building-related illness (BRI) without actually representing SBS.

Technically, nosocomial infections relate to those who are hospitalized, but health care professionals themselves may be at risk. The Center for Disease Control(CDC) publishes guidelines for control of infections[15] among hospital employees, but appropriate engineering design and maintenance can play a significant role in reducing the risks for medical professionals as well as for patients.

Natural microbial decay

Various environmental factors destroy airborne microbes.[1] Direct sunlight contains lethal levels of ultraviolet radiation. Dehydration renders most microbes inactive, although many spores may survive indefinitely. High temperatures will inactivate all pathogens, some more rapidly than others. Freezing will destroy most pathogens; except that some, especially spores, may be preserved. Oxygen slowly kills most airborne microorganisms through oxidation. Pollution levels that we tolerate our entire lives can be fatal to microorganisms. Plate-out, or adsorption, occurs on all interior building surfaces, but this removal rate tends to be negligible.

Each of these environmental processes reduces pathogen populations according to the following general equation:[1,6]

$$N = N_0 e^{-kt} \qquad (1)$$

where

N = population at time t
N_0 = population at time $t = 0$
k = rate constant for process
e = 2.718

The resulting exponential decay curve is known as a survival curve, or death curve. Often, a very small fraction of the microbial population, usually about 0.01 percent, resists chemical or physical inactivation for extended periods of exposure.[1,16]

This relation applies additively to all reduction processes—except that humidity levels will influence the effects of other factors such as ultraviolet germicidal irradiation (UVGI) and heat on a species-dependent basis. In the outdoors, sunlight, temperature extremes, and wind ensure that non-spore microbial populations decay and disperse rapidly, generally within minutes.[1,16] In the indoors, these factors

continued on page 44

continued from page 42

are controlled for human comfort, resulting in airborne microbes surviving longer, sometimes even days.[1,4]

After expulsion by sneezing or coughing, most large droplets will settle out of the air within a matter of minutes. Fig. 6 illustrates this process and is based on fitted data. Many of the micron-sized droplets will rapidly evaporate to droplet nuclei that approach the size of the individual microbe. Micron-sized particles can remain suspended for hours and spread by diffusion or air currents.[8]

Airborne microbes lose viability over time. In the absence of sunlight, the decay rates for each microbial group, based on rates measured in a variety of studies,[16] are shown in Fig. 7. Curiously, bacteria decay faster in air than viruses apparently because they depend more on moisture for their survival than do viruses.

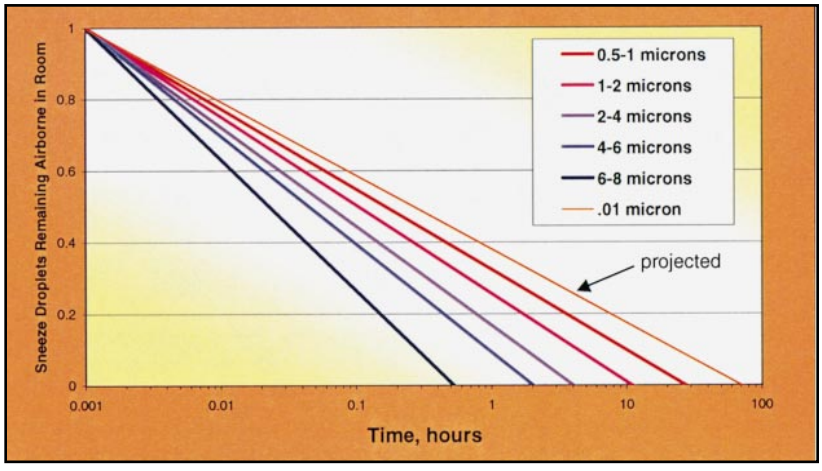

Figure 6. Disappearance of airborne sneeze droplets from room air by size. *Based on fitted, normalized data from Duguid.*[7]

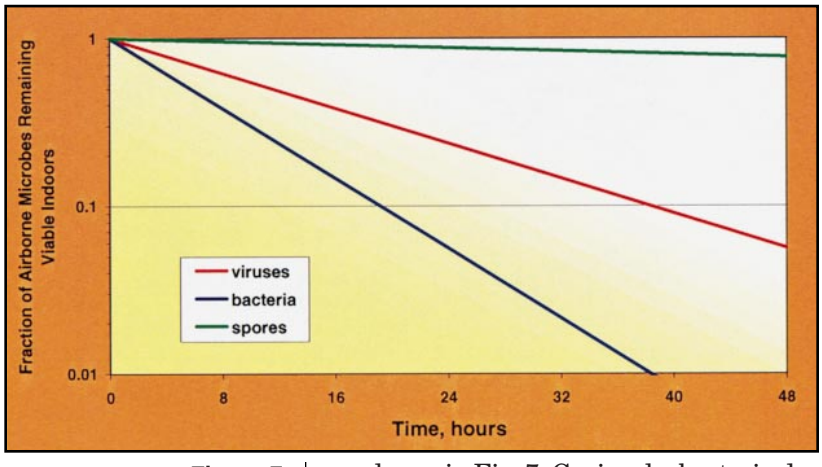

Figure 7. Viability of airborne microbes indoors in absence of sunlight. *Based on averages for each microbial group.* [1,16]

Pathways and dissemination

Fig. 8 illustrates some distinctions between airborne pathogens in relation to a typical air handling unit (AHU). Contagious viruses and bacteria come almost exclusively from humans, and they will appear only in the return air. Spores and environmental bacteria may enter from the outdoors, but once growth (amplification) occurs indoors, they may appear in the return air at higher levels than in the outdoor air. Environmental bacteria are rarely pathogenic for healthy people (Table 1), but they may provide a nutrient source for pathogenic fungi.

Spores can initially enter a building by various routes, including inlet air or infiltration, or they may be brought in with building materials, carpets, clothes, food, pets, or potting soil. In a normal, dry building, the return air will have lower levels of spores than the outdoor air,[11,12] except when snow covers the ground and outdoor spore levels approach zero. When indoor amplifiers are present, the return air could be expected to contain higher levels of spores than the outdoor air, except during dry, windy, summer conditions when outdoor levels of spores can become very high.

Once spores germinate and growth occurs in an AHU or anywhere inside the building, new spores may be generated and appear in the return air. Filters may intercept spores, but moisture may cause them to "grow through" the filter media. Cooling coils can have a pronounced filtering effect on spores,[11,12] but the presence of condensation may also cause microbial growth and amplification[10] downstream of the coils, negating the effect.

Boosting outside air flow may be an option only if the ventilation system is not the source of microbial contamination; in which case, increasing air flow may exacerbate the problem.[11] A fungus problem that is not caused by the ventilation system, such as a leaky roof or wall, requires separate remedial action such as removing the damaged material.[17]

Engineered alternatives

Natural decay mechanisms operate too slowly inside most buildings to prevent secondary infections.[16] Available engineering alternatives include purging with outside air, filtration, UVGI, and isolation through pressurization control. Each of these technologies has advantages and limitations, but optimization for any application is always possible if the microbial IAQ goals are clearly specified.

Pressurization control is commonly used in biohazard facilities and isolation rooms to prevent migration of microbes from one area to another, but inherent costs and operational instability at normal air flow rates limit feasibility for other applications.

Full outside air systems are often used in health care facilities and TB isolation rooms, subject to CDC guidelines.[15] Fig. 9 shows the effect of full purge air flow on the reduction of pathogens in a room with an initial concentration of 100 microbe CFU per cu meter. Comparing this with Fig. 10 shows the results of HEPA filtration at the same recirculation flow rates. The results are practically identical.

The use of HEPA recirculation, of course, carries a lower total energy penalty[2] in hot or cold climates. But in mild or dry climates, high percentages of outside air can prove economical, especially in applications involving evaporative coolers. Hospitals often have commitments to specific guidelines, but other facilities may select and size systems to suit their goals and budgets.

HEPA filters, for example, are not the only choice for controlling microbial IAQ. High or medium efficiency filters are capable of removing airborne pathogens, especially spores, without high operation or replacement costs.[2,16] Overall, particle removal efficiency might be improved by locating medium efficiency filters in the recirculation loop vs. the outside air intakes[16] or even downstream of the cooling coils. But, this choice will depend on each individual system's operating parameters.

Combining purge air with HEPA filtration results in performance that is essentially additive, and cost optimization becomes straightforward. Energy consumption, replacement costs, and microbial IAQ goals will dictate the economic choice for any particular installation.[16] The performance of medium efficiency filters in combination with purge air flow is not directly additive but depends on the filter efficiency vs. particle size curves, the sizes of the pathogens of concern, and the system operating parameters.

UVGI can be an efficient method to use in the right applications such as controlling microbial growth in cooling coils.[18] The continuous exposure appears to inhibit fungal growth and may kill the spores as well. In applications involving the disinfection of air streams, the effectiveness of UVGI depends on factors that include air velocity, local air flow patterns, degree of maintenance, characteristic resistance of the microbes, and humidity.[16] A single pass through a UVGI system may have a limited effect, but recirculation, either through stand-alone units or ventilation systems, will result in multiple exposures or chronic dosing.

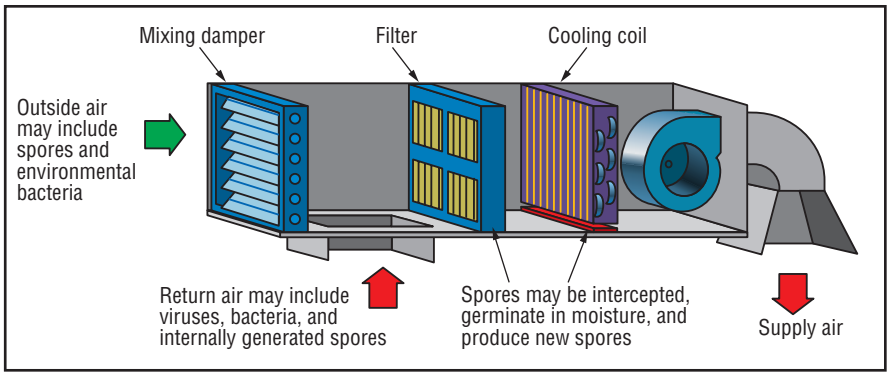

Figure 8. Sources and pathways of microbial contamination in a typical air handling unit.

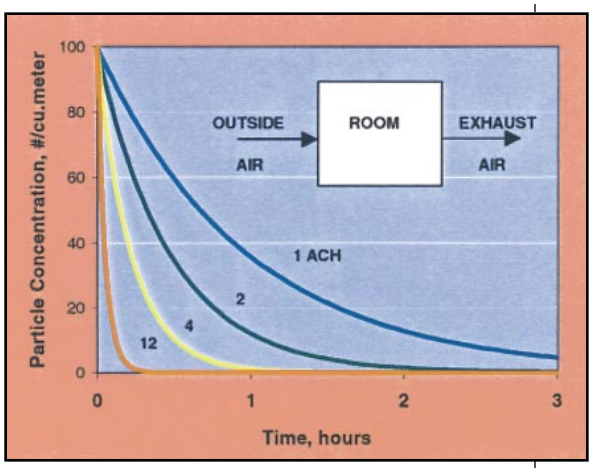

Figure 9. 100 percent outside air: effect of ach on reduction of initial level of room microbial contamination.

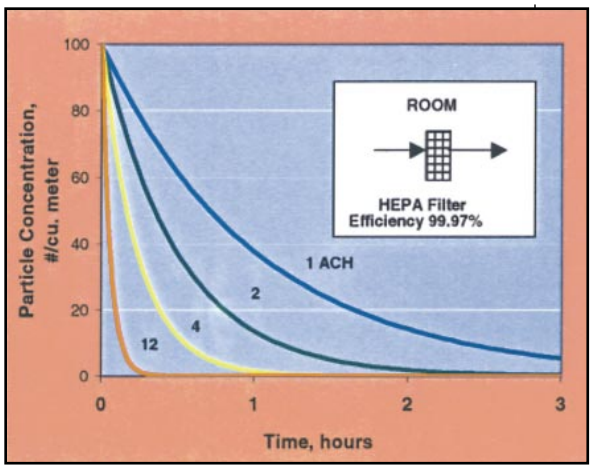

Figure 10. HEPA filter recirculation: effect of flowrate (in ach) on the reduction of initial level of room microbial contamination.

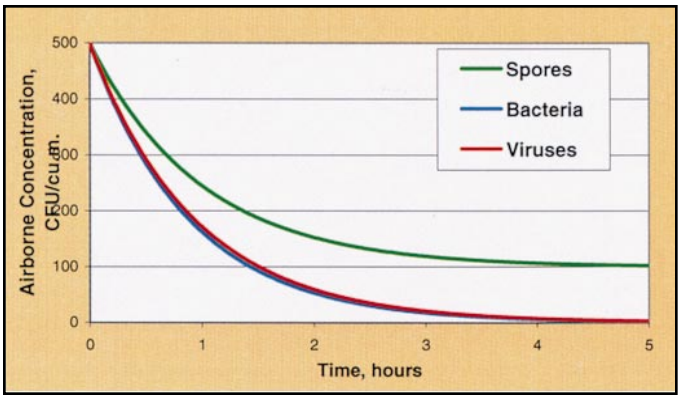

Figure 11. Effects of 25 percent outside air (1 ach) on indoor contaminant levels.[16] Outdoor spore level = 100 cfu per cu meter.

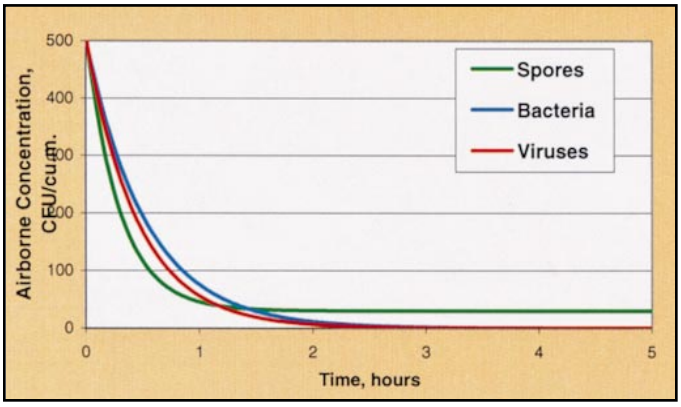

Figure 12. Effect of ASHRAE filter, 80 to 85 percent efficiency on indoor contaminant levels.[16] Recirculation with 25 percent outside air.

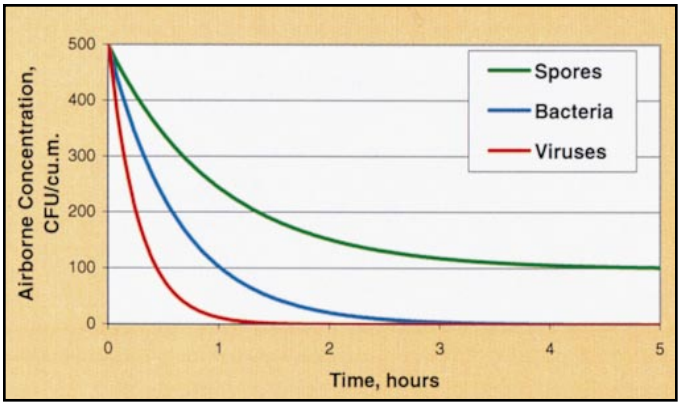

Figure 13. Effect of UVGI on indoor contaminant levels.[16] Recirculation with 25 percent OA. UVGI power mW (W=watt) per sq cm

Chronic dosing with UVGI can have a major impact on airborne viruses and bacteria.[16]

A graphic comparison of the relative effectiveness of the three main alternatives—outside air purge, filtration, and UVGI—is provided in Fig. 11 through 13. Fig. 11 shows the effect of 1 air changer per hr (ach) of outside air on reduction of room air contaminant concentrations from an initial value. Perfect mixing is assumed, along with 500 CFU per cu meter contamination of each microbial group initially, 100 CFU per cu meter of spores in the outside air, and no internal generation. Natural decay rates from Fig. 7 are incorporated in the model. The scenario of an initially contaminated room may not be realistic but provides dramatic differentiation of the effectiveness of pathogen removal.

Fig. 12 shows the effect of an ASHRAE medium efficiency filter (80 to 85 percent dust spot) to the supply air of the model building while maintaining 1 ach of outside air. The filter model describes filter efficiency vs. diameters in accordance with typical vendor performance curves.[16] Spore levels indoors are clearly reduced below outdoor ambient levels. Some reduction of bacteria and viruses can also be noted, but their removal is still dominated by the purging effect of the outside air. The filter used in this analysis provides a baseline for comparison. High efficiency filters, such as the 90 to 95 percent filters used in hospitals,[2] would result in even higher removal rates.

Fig. 13 shows the impact of a UVGI system with 25 μW (W=watt) per sq cm placed in the recirculation loop. The outside air is maintained at 1 ach, but no filters are included. Spores are relatively unaffected by the UVGI, but the viruses are markedly reduced. This model incorporates chronic dosing effects from recirculation with an exposure of 0.2 sec for each pass. The decay rate Equation 1 is applied with known rate constants[16] for a wide cross-section of the microbial species listed in Table 1.

The unusual performance characteristics of each technology have been highlighted in these examples. Inclusion of these characteristics in any evaluation, along with the IAQ design goals, ambient conditions, and internal generation rates, will dictate the choices for any given application—subject only to economic limitations.

Other alternatives

Various current or experimental technologies have the potential for reducing airborne disease transmission or indoor amplification. Biocidal filters can limit or prevent fungal growth on the filter media. Electrostatic filters *(i.e.,* electrets or electrically stimulated filters) are available but have not seen widespread

continued on page 48

continued from page 46

use. Carbon adsorbers have pore sizes too small to remove viruses, but they are effective at removing VOCs produced by some fungi and bacteria.

Other technologies currently under research include low-level ozonation, negative air ionization, and photocatalytic oxidation—a technology that may one day result in a type of light-powered, self-cleaning, microbial filter.

Conclusions

Perfect solutions to the problem of airborne disease transmission do not yet exist, but the available technologies—outside purge air, filtration, and UVGI—can be successfully implemented when their characteristic effects are understood and the goals clearly defined. Whether the application involves improvement of microbial IAQ in an office building or minimizing the risk of infection in an operating room, these technologies can be optimized individually or in combination from a cost or performance standpoint.

Finally, since microbes will never ignore opportunities provided to them, appropriate design, regular surveillance, and maintenance of these technologies in particular, and HVAC systems in general, should always be proactive. HPAC

References

1) Mitscherlich, E. and E. H. Marth. Microbial Survival in the Environment. Berlin: Springer-Verlag, 1984.

2) Burroughs, H. E. "Filtration: An investment in IAQ." HPAC Aug. 1997: 55-65.

3) Freeman, B. A. ed. Burrows Textbook of Microbiology. Philadelphia: W. B. Saunders Co., 1985.

4) Ryan, K. J. ed. Sherris Medical Microbiology. Norwalk: Appleton & Lange, 1994.

5) Mandell, G. L. ed. Principles and Practice of Infectious Diseases. New York: Wiley, 1985.

6) Hers, J. F. ed. Airborne Transmission and Airborne Infection. Proc. of VIth International Symposium on Aerobiology. Technical University at Enschede. The Netherlands: Oosthoek Publishing Company, 1973.

7) Duguid, J. P. "The size and the duration of air-carriage of respiratory droplets and droplet-nuclei." Journal of Hygiene 54 (1945): 471-479.

8) Kundsin, R. B. Architectural design and indoor microbial pollution. Oxford Press, 1988.

9) Lidwell, O. M. and R. E. O. Williams. "The epidemiology of the common cold." Journal of Hygiene 59 (1961): 309-334.

10) Woods, J. E. ed. Healthy Buildings/IAQ '97. Proc. of ASHRAE Annual IAQ Conference. Washington, DC, 1997.

11) Godish, T. Sick Buildings: Definition, Diagnosis, and Mitigation. Boca Raton: Lewis Publishers, 1995.

12) Samson, R. A., ed. Health Implications of Fungi in Indoor Environments. Amsterdam: Elsevier, 1994.

13) Burge, H. "Bioaerosols: Prevalence and health effects in the indoor environment." Journal of Allerg. Clin. Immunol. May 1990: 687-781.

14) Rao, C. Y. and H. A. Burge. "Review of quantitative standards and guidelines for fungi in indoor air." Journal of Air & Waste Management. Assoc. Sep. 1996: 899-908.

15) Castle, M. and E. Ajemian. Hospital infection control. New York: John Wiley & Sons, 1987.

16) Kowalski, W. J. "Technologies for controlling respiratory disease transmission in indoor environments: Theoretical performance and economics." Master's Thesis. Ann Arbor: UMI Dissertation Services, 1997.

17) Kemp, S. J., et al. "Filter collection efficiency and growth of microorganisms on filters loaded with outdoor air." ASHRAE Transactions Jan. 1995: 228.

18) Scheir, R. and F. B. Fencl. "Using UVC Technology to Enhance IAQ." HPAC Feb. 1996: 28-29, 85, 87.

Bibliography

1) Ahearn, D. G., et al. "Colonization by Cladosporium spp. of painted metal surfaces associated with heating and air conditioning systems." Journal of Ind. Microbiol Aug. 1991: 277-280.

2) Chang, J. C. S., K. K. Foarde and D. W. VanOsdell. "Assessment of fungal (Penicillium chrysogenum) growth on three HVAC duct materials." Environment International Apr. 1996: 425.

3) Flanningan, et al. Allergenic and toxigenic micro-organisms in houses. Pathogens in the Environment. Oxford: Blackwell Scientific Publications, 1991.

4) Hyvarinen, et al. "Influence of cooling type on airborne viable fungi." Journal of Aerosol Science 26.S1 (1995): s887-s888.

5) Macher, J. M. and J. R. Girman. "Multiplication of microorganisms in an evaporative air cooler and possible indoor air contamination." Environment International 16 (1990): 203-211.

6) Price, D. L., et al. "Colonization of fiberglass insulation used in heating, ventilation, and air conditioning systems." Journal of Ind. Microbiol. 13 (1994): 154-158.

7) Reynolds, S. J., et al. "Elevated airborne concentrations of fungi in residential and office environments." Am. Ind. Hygiene Association Journal 51 (1990): 601-604.

8) Zabel, R. A. and F. Terracina. The role of Aureobasidium pullulans in the disfigurement of latex paint films. Developments in Industrial Microbiology, Society for Industrial Microbiology, Pittsburgh, 1980.