The Application of Ultraviolet Germicidal Irradiation to Control Transmission of Airborne Disease: Bioterrorism Countermeasure

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SYNOPSIS

Bioterrorism is an area of increasing public health concern. The intent of this article is to review the air cleansing technologies available to protect building occupants from the intentional release of bioterror agents into congregate spaces (such as offices, schools, auditoriums, and transportation centers), as well as through outside air intakes and by way of recirculation air ducts. Current available technologies include increased ventilation, filtration, and ultraviolet germicidal irradiation (UVGI)

UVGI is a common tool in laboratories and health care facilities, but is not familiar to the public, or to some heating, ventilation, and air conditioning engineers. Interest in UVGI is increasing as concern about a possible malicious release of bioterror agents mounts. Recent applications of UVGI have focused on control of tuberculosis transmission, but a wide range of airborne respiratory pathogens are susceptible to deactivation by UVGI. In this article, the authors provide an overview of air disinfection technologies, and an in-depth analysis of UVGI—its history, applications, and effectiveness.

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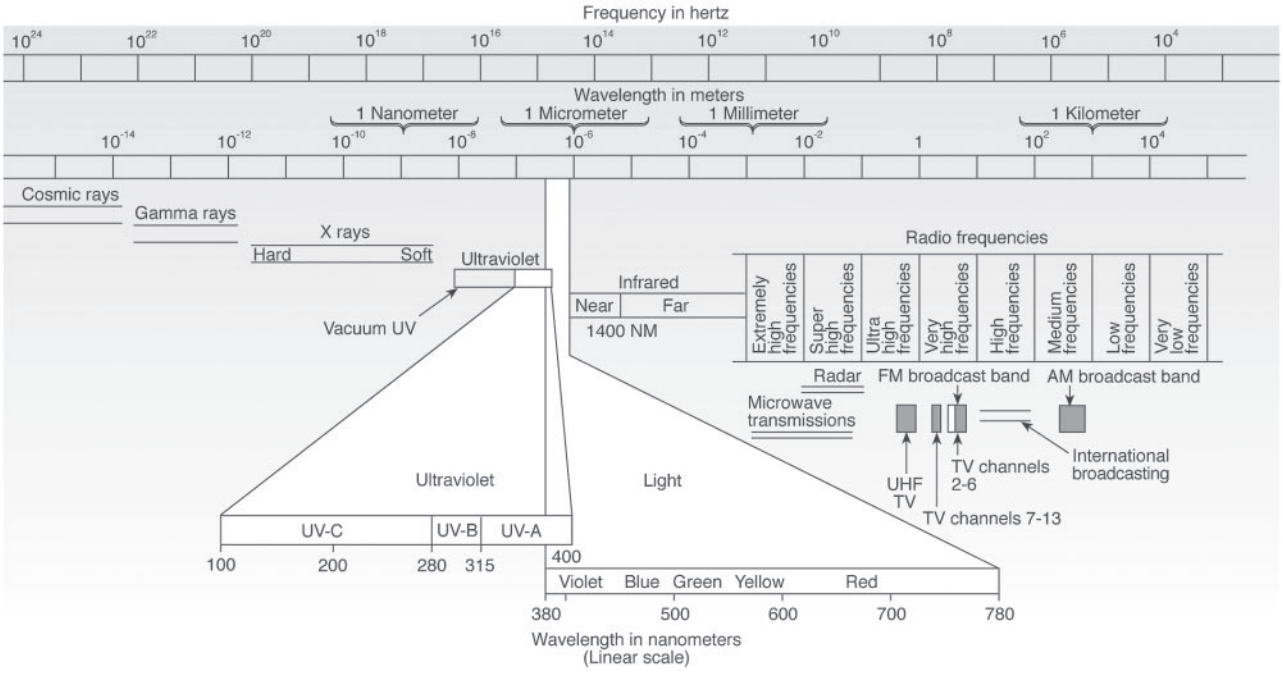
Twenty-first century bioterrorism concerns have created the need for intense review of potential countermeasures.[1–9] Our intent is to consider available technologies to protect the occupants of buildings from the intentional release of bioterror agents into indoor congregate spaces through outside air intakes and via recirculation air ducts.[10] Disinfection of air from airborne pathogens can be carried out by means of increased ventilation, filtration, and ultraviolet germicidal irradiation (UVGI). High ventilation rates of spaces occupied by people dilute and remove infectious particles (bacteria, fungi, and viruses). High efficiency particulate air (HEPA) filtration captures and retains particles small enough to be inhaled. UVGI damages the DNA of microorganisms, destroying their ability to replicate and thus rendering them non-infectious.[11–12]

Although it has long been used in laboratories and health care facilities, UVGI is the air disinfection technology least familiar to heating, ventilation, and air conditioning engineers and the public.[13–15] UVGI is produced by mercury vapor arc lamps predominately at a wavelength of 253.7 nm, within the UV-C bandwidth of the electromagnetic spectrum (Figure 1). Recent applications have focused on control of tuberculosis transmission, but a wide range of airborne respiratory pathogens are susceptible to inactivation by UVGI. Potential bioterror agents that could be aerosolized maliciously in buildings include those that cause anthrax,[5,16–19] smallpox,[20–21] viral hemorrhagic fevers,[22] pneumonic plague,[23] glanders,[24–25] tularemia,[26–27] and drug-resistant tuberculosis.[28]

Extensive laboratory and model room studies have established that the destructive effect of UVGI on bacterial and viral DNA is related to a combination of two factors: the intensity of UVGI energy to which the infectious particle is exposed, and the duration of the exposure.[15,29–33] These studies reveal a spectrum of microorganism susceptibility, dependent primarily upon the presence or absence of a cell wall and the thickness of the cell wall (see Table). Since viruses such as smallpox, influenza, and adenovirus lack a cell wall, they are more easily inactivated.[34] Common forms of vegetative bacteria are generally intermediate in susceptibility. Spores, such as *B. anthracis* in its usual state outside the body, are most difficult to penetrate by UVGI.[35,36]

As an environmental control technology designed to inactivate micro-organisms, UVGI can be installed through upper room fixtures as well as by placing UVGI lamps inside mechanical ventilation systems. UVGI offers substantial advantages over purging contaminated air by ventilation and collecting contaminants by filtration. These benefits include reduced

Figure 1. Electromagnetic spectrum illustrating UV-C in relation to other UV-bandwidths and visible light.



SOURCE: IESNA Lighting Handbook, 9th Edition; 2000.

Table. Actinic radiant exposure H at 253.7 nm necessary to inhibit colony formation in 90% (LD90) of organisms (10% survival)

Microorganism	*(H) Radiant exposure* $J \cdot m^{-2}$	*(K) Decay rate constant* $m^2 \cdot J^{-1}$	*Reference*	*Type*	*Test medium*
Bacillus anthracis	45.2	0.051	Sharp 1938[70]	Bacteria	Air
Bacillus anthracis (spores)		0.0031	Knudson 1986[36]	Bacteria	Plates
S. enteritidis	40.0	0.058	Dreyer et al. 1936[71]	Bacteria	Plates
B. megatherium sp. (veg.)	37.5*	0.061	Hercik 1937[72]	Bacteria	Plates
B. megatherium sp. (spores)	28.0	0.082	Hercik 1937[72]	Bacteria	Plates
B. paratyphosus	32.0	0.072	Dreyer et al. 1936[71]	Bacteria	Plates
B. subtilis (mixed)	71.0	0.032	Rentschler et al. 1941[73]	Bacteria	Air
	60.0	0.038	Koller 1939[74]	Bacteria	Air
B. subtilis spores	120.0	0.019	Rentschler et al. 1941[73]	Bacteria	Air
Corynebacterium diphtheriae	34.0	0.068	Sharp 1938[70]	Bacteria	Air
		0.0701	Sharp 1939[75]	Bacteria	Plates
Salmonella typhi (Eberthella typhosa)	21.4	0.108	Sharp 1938[70]	Bacteria	Air
Micrococcus candidus	60.5	0.038	Ehrismann et al. 1932[76]	Bacteria	Plates
Micrococcus piltonensis	81.0	0.028	Rentschler et al. 1941[73]	Bacteria	Air
Micrococcus sphaeroides	100.0	0.023	Rentschler et al. 1941[73]	Bacteria	Air
Neisseria catarrhalis	44.0	0.052	Rentschler et al. 1941[73]	Bacteria	Air
Agrobacterium tumefaciens (Phytomonas tumefaciens)	44.0	0.052	Rentschler et al. 1941[73]	Bacteria	Air
Proteus vulgaris	27.0	0.085	Rentschler et al. 1941[73]	Bacteria	Air
Pseudomonas aeruginosa		0.2375	Collins 1971[77]	Bacteria	Plates
		0.5721	Sharp 1940[78]	Bacteria	Air
	55.0	0.042	Ehrismann et al. 1932[76]	Bacteria	Plates
B. pyocyaneus	55.0	0.052	Ehrismann et al. 1932[76]	Bacteria	Plates
Pseudomonas fluorescens	35.0	0.066	Ehrismann et al. 1932[76]	Bacteria	
S. typhimurium	80.0	0.029	Dreyer et al. 1936[71]	Bacteria	Plates
Micrococcus luteus (Sarcina lutea)	197.0	0.012	Rentschler et al. 1941[76]	Bacteria	Air
Serratia marcescens	24.2	0.095	Rentschler et al. 1941[76]	Bacteria	Air
	22.0	0.105	Sharp 1938[62]	Bacteria	Air
	8.3	0.277	Ehrismann et al. 1932[76]	Bacteria	
		0.2208	Collins 1971[77]	Bacteria	Plates
		0.214	Riley 1976[79]	Bacteria	Air
		0.4449	Sharp 1940[78]	Bacteria	Air
Dysentery bacilli	22.0	0.105	Dreyer et al.[71]	Bacteria	Plates
Shigella paradysenteriae	16.8	0.137	Sharp 1938[76]	Bacteria	Air
Rhodospirillum rubrum (Spirillum rubrum)	44.0	0.052	Rentschler et al. 1941[73]	Bacteria	Air
Staphylococcus albus	18.4	0.125	Sharp 1938[70]	Bacteria	Air
	33.0	0.070	Rentschler et al. 1941[73]	Bacteria	Air
	18.4	0.125	Rentschler et al. 1941[73]	Bacteria	Air

(continued on p. 102)

Table (continued). Actinic radiant exposure H at 253.7 nm necessary to inhibit colony formation in 90% (LD90) of organisms (10% survival)

Microorganism	*(H) Radiant Exposure* $J \cdot m^{-2}$	*(K) Decay Rate Constant* $m^2 \cdot J^{-1}$	*Reference*	*Type*	*Test Medium*
Staphylococcus aureus	21.8	0.106	Gates 1929/1930[80]	Bacteria	Plates
	49.5	0.047	Ehrismann et al. 1932[76]	Bacteria	
		0.0886	Sharp 1939[75]	Bacteria	Plates
		0.3476	Sharp 1940[78]	Bacteria	Air
		0.0419	Abshire 1981[81]	Bacteria	Plates
		0.9602	Luckiesh 1946[15]	Bacteria	Air
Streptococcus pyogenes	26.0	0.089	Sharp 1938/39[70,75]	Bacteria	Air/Plate
(Streptococcus hemolyticus)	21.6	0.107	Sharp 1938[70]	Bacteria	Air
		0.6161	Lidwell 1950[82]	Bacteria	Plates
		0.1066	Misterlich 1984[83]	Bacteria	Air
Streptococcus lactis	61.5	0.037	Rentschler et al. 1941[73]	Bacteria	Air
Streptococcus viridans	20.0	0.115	Sharp 1938[70]	Bacteria	Air
Clostridium tetani	49.0	0.047	Sharp 1939[75]	Bacteria	Plates
Streptococcus salivarius	20.0	0.115	Sharp 1939[75]	Bacteria	Plates
Streptococcus albus	18.4	0.125	Sharp 1939[75]	Bacteria	Plates
B. prodigiosus	8.3	0.329	Ehrismann et al. 1932[76]	Bacteria	Plates
Mycobacterium tuberculosis		0.0987	David 1973[84]	Bacteria	Plates
		0.4721	Riley 1976[33]	Bacteria	Air
		0.2132	Collins 1971[77]	Bacteria	Plates
(Tubercle bacillus)	100.0	0.023	Prospeckt Philips[85]	Bacteria	Plates
Mycobacterium kansasii		0.0364	David 1973[83]	Bacteria	Air
Mycobacterium avium-intra.		0.0406	David 1973[84]	Bacteria	Air
Escherichia coli		0.0927	Sharp 1939[75]	Bacteria	Plates
		0.3759	Sharp 1940[79]	Bacteria	Air
Haemophilus influenzae		0.0599	Mongold 1992[86]	Bacteria	Plates
Adenovirus		0.0546	Jensen 1964[34]	Virus	Air
		0.0047	Rainbow 1973[87]	Virus	Plates
Vaccinia		0.1528	Jensen 1964[34]	Virus	Air
		0.1542	Galasso 1965[88]	Virus	Plates
Coxsackievirus		0.1108	Jensen 1964[34]	Virus	Air
Influenza A		0.1187	Jensen 1964[34]	Virus	Air
Cryptococcus neoformans		0.0102	Wang 1994[89]	Fungal spores	Plates
Fusarium oxysporum		0.0112	Asthana 1992[90]	Fungal spores	Plates
Fusarium solani		0.00706	Asthana 1992[90]	Fungal spores	Plates
Penicillium italicum		0.01259	Asthana 1992[90]	Fungal spores	Plates
Penicillium digitatum		0.00718	Asthana 1992[90]	Fungal spores	Plates
Rhizopus nigricans spores		0.00861	Luckiesh 1946[15]	Fungal spores	Air
Cladosporium herbarum		0.00370	Luckiesh 1946[15]	Fungal spores	Air
Scopulariopsis brevicaulis		0.00344	Luckiesh 1946[15]	Fungal spores	Air
Mucor mucedo		0.00399	Luckiesh 1946[15]	Fungal spores	Air
Penicillium chrysogenum		0.00434	Luckiesh 1946[15]	Fungal spores	Air
Aspergillus amstelodami		0.00344	Luckiesh 1946[15]	Fungal spores	Air

Tabular information adapted from CIE[53] and Penn State University Aerobiology.[59]

NOTE: Although data from both air and surface (plate) exposures are intermixed in this table, the LD_{90} doses for each cannot be compared directly. It is generally much easier to inactivate microbes in the air than on surfaces. In both air and on surfaces the LD_{90} depends on the exact conditions of each experiment. Susceptibility differences in air between species may reflect differences in the conditions of the study as well as differences proper to the species.

cost, ease of installation and maintenance, and potential effectiveness when used in congregate settings.[37,38] Applications include commercial and government office buildings, health care institutions, schools, dormitories and barracks, indoor shopping malls, and public transportation facilities, including airplanes.

DISEASE TRANSMISSION THROUGH INHALATION OF DROPLET NUCLEI

Transmission of airborne disease can be understood as a function of the concentration of respirable infectious particles in air. Such particles are called droplet nuclei.[39] On average, droplet nuclei are about 3 µm (micrometers) in diameter and, when inhaled, are capable of bypassing the protective mechanisms of the upper respiratory tract and causing infection. Droplet nuclei are thus responsible for human-to-human transmission of many airborne infectious diseases. When a contagious individual coughs or sneezes, sputum droplets containing infectious particles (bacteria, viruses) are released. The larger ones fall to the floor where they adhere to surfaces and dust particles, and are no longer infectious. Smaller particles remain airborne long enough that the moist coating of saliva and mucus evaporates, leaving a residual dry *nucleus* of the droplet that may include one or more bacteria or viruses.

Inhalation of a single droplet nucleus may be capable of initiating pulmonary tuberculosis in highly susceptible hosts, individuals with AIDS, for instance, whereas more resistant hosts may require larger infectious doses. This implies that there is no specific threshold air concentration below which transmission will not occur. Some bioterrorism agents can be aerosolized and maliciously introduced into congregate settings. Such attacks would present problems for air disinfection technologies that are both similar to and different from those presented by person-to-person transmission. The issues are similar because the control principles are essentially the same, but they differ because the concentrations of infectious agents may be much higher than ordinarily seen with natural infections under usual circumstances.

AIR DISINFECTION TECHNOLOGIES

Building ventilation and directional airflow as protection against airborne infection

A standard engineering approach to the control of airborne infection inside buildings consists of ventilation and directional airflow. Hospital isolation rooms, for example, employ high rates of ventilation to dilute and remove infectious particles, and directional airflow to prevent them from entering corridors or adjacent rooms. Building codes mandate a range of ventilation rates for various public access buildings, requiring higher rates for schools, for instance, than for department stores. Building ventilation is quantified both as outdoor air volume per unit time per person, and as room air changes per hour (ACH), irrespective of occupancy. After the volume of air entering a room equals the volume of the room, one room air exchange is said to have taken place. In old buildings, natural ventilation occurs through open windows and building leaks. Natural ventilation rates range from as little as one-fourth of an air exchange per hour (0.25 ACH) in a very tightly constructed building to several air exchanges per hour in one less tightly constructed.

Public buildings in industrialized countries are provided with mechanical HVAC systems that usually condition and recirculate most of the returned air, exhausting some and replacing it with outside air to control odors, CO_2 build-up, and air contaminants such as smoke.[40] Because developers, architects, and engineers are most familiar with these technologies, it is understandable that increased ventilation is often proposed to reduce airborne disease transmission in buildings.[11] We will make the case that ventilation is neither the only nor the best method of air disinfection for all airborne threats, including those posed by bioterrorism.

Building ventilation

With a perfectly uniform concentration of particles and uniform mixing of incoming air, 63% of the air and airborne organisms will be removed with each air change.[41] However, under more realistic conditions, when there is an uneven distribution of infectious particles and uneven mixing of fresh air with contaminated air, less than 63% of air and airborne particles are flushed out with each air change.[41] The true decreases per air change that have been measured are in the range of 20% to 60%.[42]

Another consequence of the uneven distribution of infectious particles in air is that some exposed individuals may inhale multiple infectious doses while others inhale none during the same time period. Mathematical models of airborne infection have been developed to describe the impact of ventilation on the transmission of airborne infectious diseases.[39,43–45] (See Appendix 1 for an example of applying such a model.)

Building ventilation is often limited by design (capacity of blowers, ducts), comfort (noise, drafts), and by economic consideration (cost of conditioning out-

side air). Whereas an isolation room or intensive care unit may be designed with 12 or more air changes per hour, many public indoor spaces are not. For these reasons, in public indoor spaces where airborne transmission is likely, it is desirable to consider supplementing ventilation with other means of air disinfection, such as air filtration or UVGI. The air-disinfecting effects of these measures have been equated to ventilation *for air disinfection purposes only,* and termed "equivalent ventilation." That is, when 63% of airborne infectious particles are removed by filtration or inactivated by UVGI, they have produced one "equivalent air change." Particle filters and UVGI do not remove CO_2 or replace O_2, so "equivalency" is limited to air disinfection. Ventilation is still required to serve its usual functions.

Air disinfection by filtration or UVGI follows the same logarithmic clearance relationship described for ventilation. This is a fundamental relationship for all disinfecting processes where a certain percentage of a population of organisms is inactivated with each exposure. One well-mixed air change (produced by ventilation, filtration, or UVGI) inactivates approximately 63% of airborne organisms, a second air change inactivates approximately 63% of the remainder, and so on, producing a logarithmic decay curve. However, because filtered air is recirculated, it may be possible to achieve higher levels of equivalent air changes with filtration at lower cost than by means of outdoor air ventilation, because heating and cooling costs are less. Like ventilation, air filtration requires mechanical airflow and may also be limited by occupant comfort (noise and drafts). UVGI depends on the movement of contaminated lower room air into the irradiated upper room where organisms can be rapidly inactivated. Although low velocity paddle fans have been used to increase air mixing between the lower and upper room, even passive air mixing has been shown to produce useful numbers of equivalent room air changes inexpensively, and without noise or drafts.[32,33] For some applications, therefore, UVGI may disinfect air equivalently to high levels of ventilation, but at lower installation and operating costs. Air disinfection in place is especially important for dealing with an unsuspected infectious source, a person, or possibly an attack, where isolation and individual respiratory protection may not be an option.

Air filtration

High efficiency particulate air (HEPA) filters remove over 99.99% of airborne particles that arrive at the filter media.[46] HEPA filters, often used for infection control, are tested with an aerosol containing monodispersed particles 0.3 µm in diameter, the most penetrating aerodynamic particle size. Filtered recirculated air can be substituted for a portion of outside air, avoiding the cost of heating, cooling, and dehumidifying outside air. Filter technology is used extensively on airliners, where the cost of heating frigid outdoor air at altitude is high. However, as with UV disinfection, HEPA filtration neither removes carbon dioxide nor adds oxygen, so that adequate outdoor ventilation for these purposes is always necessary. The limitations of HEPA filtration for air disinfection are similar to those of increasing building ventilation rates to control transmission of infections diseases, i.e., the need for high levels of progressively less efficient air changes. Also, HEPA filters generate resistance to airflow, necessitating more powerful fans that produce noise and vibration. Filters are costly and must be changed periodically. To be fully effective, HEPA filtration systems must be leakproof. They require careful routine maintenance,[10] as do all systems that are counted on to perform a critical function. Another limitation particular to portable air filtration units is the potential to reentrain already filtered air because the unit's intake and exhaust locations are usually necessarily close to one another. This short-circuiting reduces the efficiency of air disinfection by filtration.

Ultraviolet germicidal irradiation (UVGI)

History of UVGI research. Disinfection of air in the upper part of rooms using ultraviolet energy has been studied as a public health strategy to control transmission of airborne disease since investigations by William Firth Wells in the 1930s at the Harvard School of Public Health[47,48] Dr. Richard Riley and colleagues advanced this work at Johns Hopkins Medical School with a focus on tuberculosis control.[49,50]

Streptomycin, the first of a series of anti-tuberculosis antibiotics, became available in 1946, followed by isonicotinic acid hydrazide (INH) in the 1960s. It came to be generally believed that the TB problem would be solved by antibiotic therapy, and enthusiasm for ultraviolet air disinfection waned. The sanitarium movement ended in the same period. But in 1985, contrary to predictions, the United States national TB case rate increased for the first time in the 20th century, fueled in part by transmission in homeless shelters, prisons, jails, hospitals, and other congregate settings. One of the results of this upswing in TB transmission was renewed interest in the potential value of UVGI to prevent TB transmission.

Although United States TB case rates are once again in decline, there is growing concern about the spread of the disease, including multidrug resistant strains, in

many parts of the world, especially in sub-Saharan Africa, parts of Asia, and in the former Soviet Union. Transmission in hospitals, prisons, refugee camps, and other congregate settings is of greater concern, and practical methods for protecting building occupants are needed. The Tuberculosis Ultraviolet Shelter Study (TUSS), a large clinical trial of the efficacy of UVGI to reduce TB transmission in congregate settings, is being conducted from Saint Vincent's Hospital in New York and the Harvard School of Public Health.[51] The threat of bioterrorism (including multidrug-resistant TB) in this country is a significant reason to reconsider the application of UVGI.

Experimental evidence of UVGI effectiveness. The scientific development of UVGI began with research on the intrinsic susceptibility of microorganisms to ultraviolet energy. Ample experimental data exist concerning the quantity of UVGI energy needed to inactivate microorganisms in the air, on surfaces, and in water.[52] Our discussion is concerned only with UVGI applications for air disinfection. Laboratory and model room experiments have compared the clearance rate of microorganisms by UVGI alone to the number of additional room air changes required to produce the same effect. Both early and recent studies indicate that properly installed UVGI lamps in the upper portion of a normally ventilated room can disinfect air of aerosolized mycobacteria in the range of 10 to 20 ACH equivalents.[33,41]

Species susceptibility to UVGI

The range of susceptibility for bacteria extends from *streptococcus* species, the most susceptible; to *Mycobacterium tuberculosis* (MTb), the agent for tuberculosis, moderately susceptible; to the spore forms of bacteria (such as anthrax outside the human body), the least susceptible. Although effective UVGI doses have been established for a wide range of microbial species, many of these doses were determined for organisms on surfaces rather than in aerosolized form (see Table). Thus the results of these studies may overestimate the dose needed, because it is far easier to inactivate airborne organisms than those bound to surfaces. MTb is both moderately susceptible to UVGI and a significant human pathogen, and therefore has often been used as a reference organism when UVGI exposure requirements are determined for individual pathogens. For example, a dose of UVGI that inactivates MTb will be more than adequate to inactivate respiratory viruses, such as measles, influenza, and presumably smallpox.

To destroy all the infectious particles in a quantum of airborne organisms by UVGI exposure requires a much higher dose than to destroy, for example, 90% of the pathogens, in part because of biological variation in susceptibility. For each microbial species, an experimental dose-response curve varies with environmental exposure conditions such as humidity, temperature, and particle size. Fractional survival of bacteria exposed to UVGI is described in a mathematical expression detailed in Appendix 2. This expression is used to estimate UVGI effectiveness for the destruction of specific airborne infectious organisms in a range of ventilation and UV energy settings.

To summarize, current data indicate that the incident UVGI radiant quantity required to inactivate infectious particles relates to the:

- Microbial species and its ability to recover from damage induced by UV radiation;
- Presence of sufficiently high radiant exposure dose over time;
- Degree of relative humidity.

Accurate data on the impact of these factors are essential for planning UV air disinfection, but exist for only a limited number of species of interest for naturally transmitted infection, and for even fewer agents with bioterrorism potential.

To develop practical application of UVGI in high-risk settings, Riley conducted bench-scale studies during which he exposed both virulent and non-virulent bacillus Calmette-Guérin (BCG), tubercle bacilli, and other organisms to UVGI energy of known intensity and duration under conditions of controlled temperature and humidity.[33] These studies demonstrated a 90% lethal dose (LD_{90}) for virulent TB and for BCG of 12 seconds exposure at 50 $\mu W/cm^2$, or 60 seconds at 10 $\mu W/cm^2$.[33] It is relatively easy in practice to produce UVGI intensity in the 10 to 50-$\mu W/cm^2$ range in the upper room using available UVGI fixtures and lamps. Because rapid overexposure could occur at these intensities, guidelines for maintenance and safe operation of UVGI systems require deactivation when people are at work in the overhead disinfection zone. From these experiments, Riley and other investigators began testing the effectiveness of UVGI in model rooms where neither the average dose nor duration of exposure for test organisms could be estimated a priori.

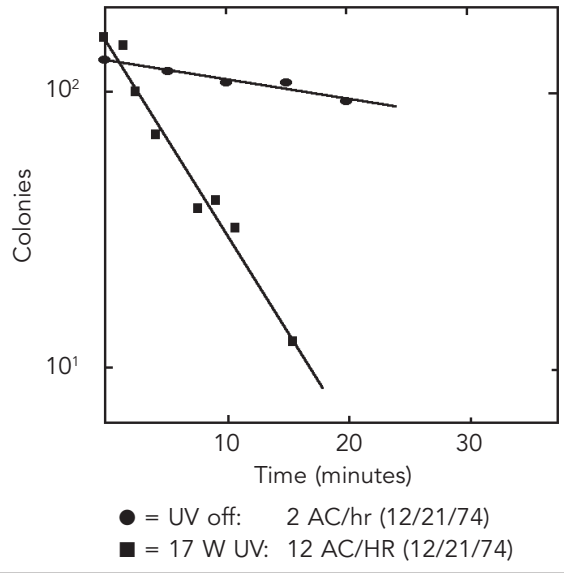
Model room studies

Evidence that upper room UVGI energy provides useful air disinfection is derived from experiments in model rooms.[30,32,53–55] Test organisms have been aerosolized into these spaces and their disappearance rates with and without UVGI measured by quantitative air sampling. A characteristic study used BCG aerosolized

into a naturally ventilated 18.6 m^2 (200 ft^2) room.[33] A single 17-watt UVGI lamp irradiating the upper part of the room added the equivalent of 10 ACH to the ambient ventilation of two ACH. Two UVGI lamps producing a total of 46 watts irradiating the upper room added the equivalent of 33 ACH to the ambient ventilation of 4 ACH (see Figure 2).

Model room experiments with other test organisms have shown that air disinfection in the lower room can be improved by increasing the dose of UVGI in the upper room.[53] This effect has been amplified further by increasing air mixing, either through the use of fans or by increasing temperature gradients between the upper and lower room, which also improves air flow. Air mixing is essential for effectiveness of UVGI air disinfection, because the infectious particles must be transported to the irradiated zone.[32,56,57] A source of heat in the room increases convection currents and air mixing. It should be noted here that heat flows from an adult human being at the equivalence of a 100-watt incandescent light bulb.[58] This finding is based on studies revealing that humans emit 400 BTU per hour during light activity, a well-established figure from physiologic studies, and that an incandescent bulb emits 3.41 BTU-per-watt rating.

Figure 2. Disappearance of aerosolized bacillus Calmette-Guérin (BCG) from room air with and without upper room ultraviolet (UVGI) irradiation using one suspended fixture with one 17 W lamp.



AC/hr = air changes/hour

Y-axis = viable colonies remaining in air

X-axis = duration of exposure to UVGI

SOURCE: adapted from Riley.[34]

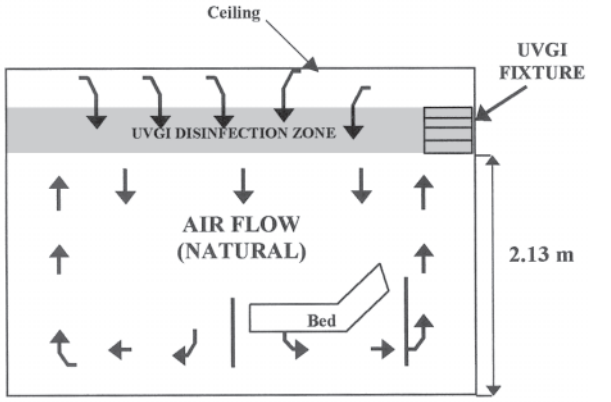
APPLYING UVGI

Upper room application of UVGI in congregate settings

The rationale of UVGI applications is that germicidal irradiation (UVGI, 253.7 nm) placed in the upper part of occupied spaces will safely and effectively interrupt the transmission of certain airborne human infectious disorders, e.g., such common diseases as influenza, adenovirus infection, measles, and tuberculosis. Depending on the UV susceptibility of the organisms and the mode of transmission, agents of potential bioterror concern are assumed to respond in a similar way. Inactivation of microorganisms occurs when they reach the UVGI zone. Vigorous upflow of air rapidly brings infectious particles into the upper room. The more vigorous the upflow, the shorter the stay of the particles per pass within the zone, but the more passes per unit time. For a fixed UVGI intensity there is a theoretical optimal duration of UVGI exposure that will maximize the inactivation of organisms in a room. The optimal duration can be computed from knowledge of UVGI lamp (bulb) energy output, fixture (lamp holder) configuration and placement, room geometry, and ventilation/air circulation patterns. In practice, this computation is seldom made because the duration of exposure required for lethal effect using current UVGI technology is so short that room air mixing is the rate-limiting factor.

UVGI systems are designed so that fixtures generate a controlled zone (Figure 3) of radiation in the space well above occupant's heads. Fixtures are distributed in rooms to provide coverage of as much of the below-ceiling area as is practical. Infectious particles are brought into the UVGI beam by air currents

Figure 3. Section view of wall-mounted UVGI fixture irradiating the upper room space over a hospital bed



Arrows indicate convection currents.

generated by body heat, ventilation systems, occupant motion, fans, and other factors. When infectious particles enter the beam, UVGI energy damages the DNA, interfering with replication, and thereby rendering the microorganism noninfective.

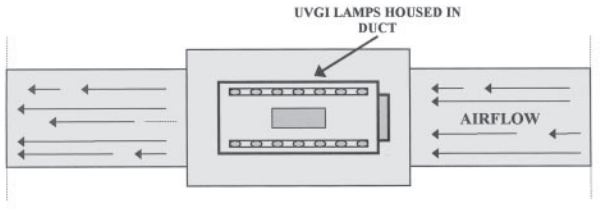
The application of UVGI faces practical limits related to old building structures, but new construction can be planned to accommodate UVGI placement. For example, building plans can be modified to adjust floor-to-ceiling height and to optimize space utilization so that UVGI can be installed in conjunction with HVAC designs. These factors demand careful planning of UVGI fixture placement in relation to room geometry and ventilation mode to secure maximum inactivation of infectious agents. This must be achieved while maintaining safety in concordance with daily threshold limitation values for human UVGI exposure.

As noted, the effectiveness of UVGI can be compared to ventilation in terms of equivalent air changes, and can be estimated, although not readily measured for each application, as exposure of test organisms requires. As an example, in a room normally ventilated by six air exchanges per hour, adding an upper room UVGI system might achieve the air-cleansing equivalent of approximately an additional 10 to 20 ACH.

A model of upper room UV air disinfection has been constructed that takes into account both air mixing and upper room inactivation of organisms, based on experimental data. For the purposes of this discussion, we will assume that a minimum number of 10 lower room air volumes pass through the upper room UVGI exposure zone per hour. With the following assumptions, the required exposure time for inactivation of an infectious airborne particle can be calculated: (*a*) the room height is 2.4 m (8 ft.); (*b*) the lower 1.2 m (4 ft.) of air circulate vertically through the upper 1.2 m (4 ft.); (*c*) the average height from which the lower 1.2 m (4 ft.) of air rises is two feet above the floor (the midpoint of the lower 1.2 m [4 ft.] air layer); (*d*) an amount of air equivalent to 20 times the lower room volume circulates through the upper room each hour (to provide the equivalent of 10 complete upper room volume air changes); (*e*) the irradiation zone is the upper 0.7 m (2 ft.) of the room.

Based on these assumptions, air travels 1.8 m (6 ft.) up and 1.8 m (6 ft.) down 20 times per hour, or at the velocity of 1.2 m (4 ft.) per minute. Air is in the UVGI zone for two feet during travel up and two feet during travel down for each air turnover through the upper zone. For infectious particles in the air, this is the equivalent of a one-minute exposure to a minimum of 10 $\mu W/cm^2$ of UVGI, an exposure time-intensity product that corresponds to Riley's experimentally determined dose for 90% kill rate. This calculation is conservative in that it neglects horizontal travel of air within the upper 0.7 m (2 ft.), the irradiation zone.

Figure 4. Placement of UVGI lamps in air ductwork (section view) with UVGI lamps placed perpendicular to the airflow



UVGI in-duct systems

TB control guidelines recommend the use of the in-duct systems strategy for disinfection of air (see Figure 4).[11] Currently, however, no public or private database exists for designing applications of UVGI within air ducts and ensuring "kill" rates adequate for the proposed use. Several recent studies have developed methods to predict the rate of air stream disinfection produced by in-duct UVGI systems.[59,60] These studies allow better understanding of the physical location of UVGI lamps (whether in the supply duct near the coil or in the return duct near the filter) and the radiation densities required, given the multiple reflections that occur within the duct when common ductwork materials are used.[59] Ongoing studies are being conducted by the heating, ventilating, air conditioning, and refrigeration industry to map UVGI intensity distribution within ductwork, UV lamp and ballast characteristics, air velocity impact on time in the "kill" zone, and the impact of temperature and humidity on effectiveness.[61] Other factors under study include the susceptibility of microorganisms to UVGI coupled with photocatalytic in-duct coatings used to remove air contaminants. While much information exists, it is not as yet integrated into a whole building systems approach.

TECHNICAL OBJECTIVES

Engineering aspects of UVGI

For most purposes, upper room UVGI is applied using long-established guidelines based on early experiments. Usually no attempt is made to quantify either the equivalent air changes produced or the contribution

of ventilation to air mixing. However, these measurements can be made for research purposes and for critical applications using a mathematical UV effectiveness index.[62] A detailed evaluation of UVGI air disinfection requires qualitative and quantitative descriptions of the ventilation systems in buildings. This information is obtained by on-site measurements of air change rates and air mixing factors determined from tracer gas techniques. If mechanical ventilation is present, exhaust and supply airflow is measured using a flowhood, such as an Alnor Balometer, room dimensions and population density, photographs, and other relevant descriptors. The influence of open windows and doors on interior airflow patterns must also be evaluated with respect to air change ratios, mixing factors, and UVGI exposure intensity and duration. Although the latter will change with climatic conditions, so will mechanical ventilation conditions, for example, between heating and cooling periods of the year.

Upper room UVGI could be more effective if it were feasible simply to flood the space with very high levels of UVGI energy. However, this cannot be done because of human safety issues related to external eye irritation (keratoconjunctivitis) and skin erythema. UVGI fixtures are designed for maximum upper room irradiation while limiting exposure to room occupants. Current fixture designs rely on deep louvers to prevent overexposure at eye level or excessive reflection from low ceilings. However, louvers absorb a large fraction of potentially useful UV energy, rendering current UVGI fixtures inefficient. In the future, more efficient fixture designs are likely to utilize precise parabolic reflectors to direct UV beams from powerful, pencil-thin sources.

Among the factors that determine UVGI fixture placement are the characteristics of the ventilation system, safety considerations, occupancy patterns, existing structural limitations, cost, and maintenance. Placement of UVGI fixtures is not practical in some spaces. A minimum ceiling height of 2.4 m (8 ft.) is required to assure that direct UV irradiation is above eye level. The higher the ceilings, the better for UVGI air disinfection purposes, from both the safety and efficacy perspectives. For spaces with lower ceilings, UVGI duct irradiation devices must be employed.[11] Also, there may be features of a room or its furnishings, such as podia or bunk beds, that can effectively raise the eye level of occupants above 1.8 m (6 ft.). UVGI applications inside of air ducts may be particularly appropriate in such spaces.

Practical and safe use of UVGI energy sources

Human safety is always the prime consideration, and modern UV systems are designed to recognize this concern. Two side effects are known to occur following human overexposure to UV-C energy. These are skin reddening (erythema, akin to sunburn) and photokeratitis (external eye inflammation).

UVGI lamps emit 90% of their radiant energy as UV-C at 254 nm and 10% at other UV and visible wavelengths. UV-C is "short-wave" radiation, almost totally absorbed by the stratum corneum of the skin. Therefore, it does not produce a significant degree of sunburn even after inadvertent extensive skin exposure, although it can cause a mild erythema. However, the cornea of the eye is very sensitive to UV-C and, with overexposure, a painful photokeratitis can occur. This effect is well known, for instance, among arc welders not properly protected. UV photokeratitis clears in 24 to 48 hours, without sequellae. UV-C energy does not penetrate the cornea, and therefore adverse effects on the lens and retina are not possible in real-life scenarios, even for those who have had their eye lenses removed during cataract surgery (aphakia). In consideration of safety issues, it is important to remember that human exposure to UV from sunlight when outdoors is several orders of magnitude greater than exposure indoors from upper room UV in the lower part of the room. Sunlight contains longer wavelength UVA and UVB, known to be responsible for skin cancer and some forms of cataracts[63,64] (Personal communication, DH Sliney, October 3, 2002).

Correctly installed UVGI does not produce photokeratitis or skin erythema. The Tuberculosis Ultraviolet Shelter Study (TUSS), for example, has placed UVGI in 12 shelters in five cities, and has noted no eye or skin complaints since TUSS started in 1997, despite periodic questionnaires soliciting such symptoms.

In current UVGI applications, the fixtures are placed well above eye level (>2m). Thus there exists little potential of UV overexposure for room occupants. Maintenance staff who may need to work in the upper portion of the space (such as for painting the ceiling or changing bulbs) must be trained to turn off the UV system during that time (and then must remember to turn it on again!).

We recommend UVGI fixtures designed to contain UVGI lamps that produce intensities of at least 50 $\mu W/cm^2$ at a 1 m (3 ft.) distance and at least 10 $\mu W/cm^2$ (centerline) at about 3 m (10 ft.) from the UVGI lamp. Depending on the room configuration, wall-mounted fixtures containing one or two 17-watt lamps per 20 m^2 (200 $ft.^2$) of ceiling space are usually appro-

priate. Suspended fixtures are often used for larger areas with high ceilings because wall-mounted fixtures may be too distant from the central air space. Some suspended fixtures contain lamps that distribute UVGI horizontally over 360 degrees.

The American Conference of Governmental Industrial Hygienists (ACGIH) has issued guidelines on the safety exposure limits for UV-C that are currently used by the National Institutes for Occupational Safety and Health (NIOSH).[65] ACGIH recommends threshold limit values (TLVs®), under which most persons can work consistently for eight-hour periods without adverse effects. The TLV® for UV-C exposure is 6.0 mJ/cm^2 for an eight-hour exposure. Exposure above 6.0 mJ/cm^2 during an eight-hour period may result in erythema of the skin and photokeratitis. Despite this safety threshold, estimating the actual exposure of room occupants is not simple. If eye and skin exposure were uniform and continuous, one would reach a total dose of 6.0 mJ/cm^2 at an irradiance of 0.2 μW/cm^2. For many years, this value was inappropriately applied as an upper limit for UVGI intensity at eye level in rooms where upper room UVGI was in use. In reality, however, blinking of eyelids, shading from brows, turning of the head, and numerous other factors normally limit human eye exposure to the maximum irradiance in the lower room with UVGI to a small fraction of the time that a person spends in the room. These same factors normally protect human beings out of doors from photokeratitis due to sunlight exposure.

Louvers on many current commercial UVGI fixtures are designed to prevent exposure of more than 0.2 μW/cm^2 at eye level. This design, however, reduces UVGI output and sacrifices upper room efficacy in order to achieve unnecessarily low levels in the lower room. Personal monitoring of UV exposure using miniature UV meters has shown that individuals occupying rooms with eye level irradiance several fold in excess of 0.2 μW/cm^2 actually receive only a small fraction of the eight-hour TLV®.[66]

Where should UVGI be applied today?

UVGI should be considered as a component of emergency preparedness plans for existing public buildings and a wide range of congregate settings. Illustrations of UVGI application are shown in an excerpt from an architectural plan (Figures 5a and 5b). All of these areas could be covered by commercially available upper room UVGI equipment, and would be more practical for air cleansing than installation of costly filtration or separate air handling systems. Guidelines for upper room UVGI placement have been published.[37,38] Less well understood is where to place UVGI lamps within ductwork. CDC 1994 Guidelines for TB control can be used as a starting point.[33]

WHAT WORK NEEDS TO BE DONE?

An unrecognized opportunity exists to integrate UVGI as a subsystem within existing buildings and those under design post–September 11, 2001. Upper room UVGI can be easily installed in vulnerable areas within buildings at a small fraction of the cost of installing high efficiency filtration. Additionally, there is a need to establish quality control measures to assure that each of the strategies (filtration, dilutional ventilation, and UVGI) function as intended. These measures require an initial commissioning process and continued maintenance and monitoring. Training will be necessary to bring design and application tools to developers, engineers, and architects.

Bioterrorism concerns have caused an enhanced interest in the development of UVGI in indoor public spaces and large buildings. Additional studies will focus on UVGI efficacy in defined structures, definitive testing for the UVGI dose required to inactivate a wide range of specific pathogens, dosimetry, personal UVGI monitors, standardization of methods to test UVGI system components (lamps, fixtures, ballasts), and the development of design guidelines and computer programs for UVGI applications in high-risk settings.

The integration of ventilation, filtration, and UVGI technologies is the wise approach to airborne disease mitigation for entire buildings, based on the range of infectious agents to be neutralized. The final intention is to achieve energy-efficient and biologically effective control. Basic principles are understood and now require development into guidelines for manufacturers, developers, architects, and engineers.

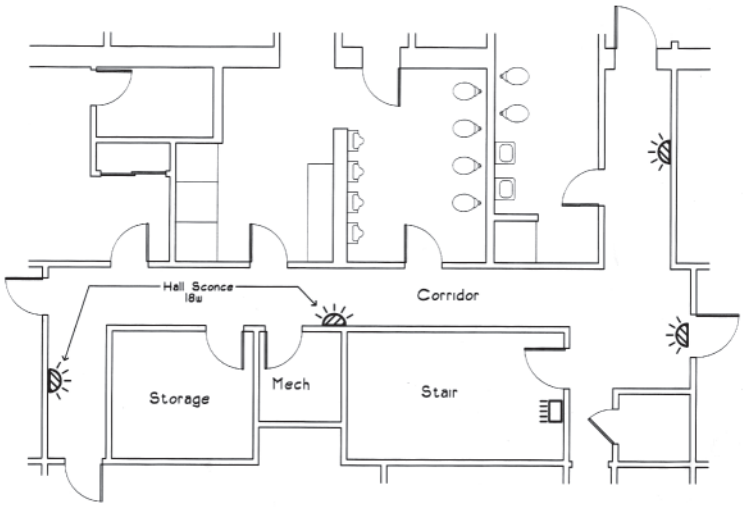
APPENDIX 1. APPLICATION OF INCIDENCE OF INFECTION MODELS

In these models, the incidence of infection (λ) at time t is a function of the prevalence (Pr) of infectious cases at time t, the average pulmonary ventilation rate per person (p), the duration of the exposure (d), the outdoor air ventilation rate (v), and the number of doses of airborne infection added to the air per unit time by each infectious person (q).

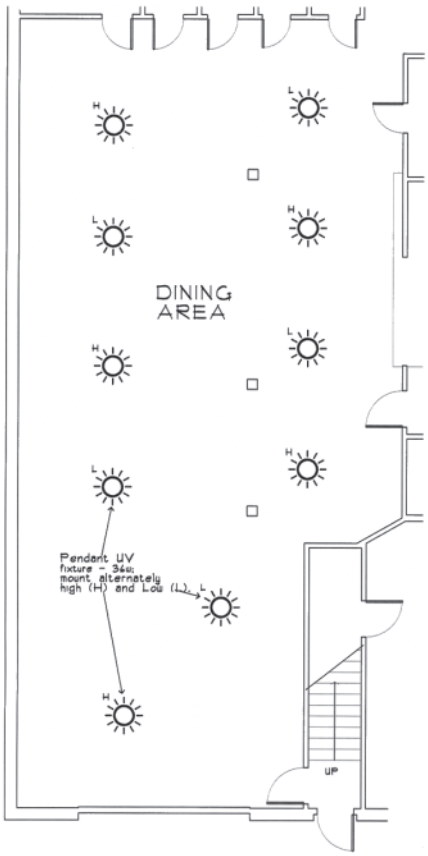
Assuming that the number of infectious cases is constant, the cumulative incidence (CI) of infection is:

$$CI = S(1 - e^{-\lambda t}) = S(1 - e^{-\Pr \cdot q \cdot p \cdot d/v})$$

Figures 5a and 5b. UVGI fixture placement for circulation corridors and congregate settings (dining area) in plan view



5a



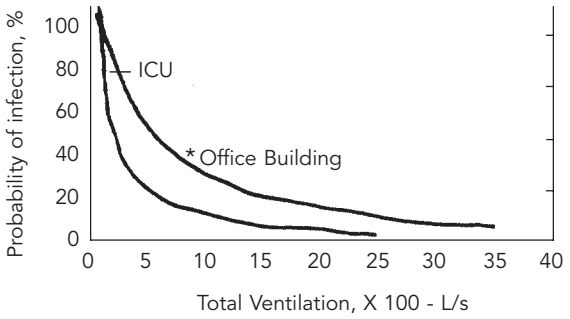
5b

In this expression, the incidence rate (λ) is equivalent to the total number of doses per unit volume of air per unit of time. Note that the terminology used in this expression, the Wells-Riley equation, reflects current epidemiology convention and differs slightly from that used to describe the probability of airborne infection in earlier publications.[41,67] Dividing through the equation by *S* (susceptibles) yields an expression for the fraction infected among those exposed, or the probability of infection, $(1 - e^{-\Pr \cdot q \cdot p \cdot d/v})$. Plotting the probability of infection as function of ventilation with outside air (v) in volume per unit time for various values of Pr, *q, p,* or *d,* generates a family of logarithmic decay curves.

Figure 6 shows two examples of such curves derived from two actual tuberculosis exposures.[68,69] The two labeled points, "ICU" and "Office building" reflect the probability of infection $(1 - e^{-\Pr \cdot q \cdot p \cdot d/v})$ at the actual ventilation rate (*v*) for each exposure, as indicated in the figure. Assuming that all other factors (Pr, *q, p, d,* and *S*) remain constant, the curves represent the theoretical probability of infection predicted for increasing or decreasing ventilation (*v*) above or below the actual values.

Although plotted on the same axes, the curves cannot be compared directly to one another because the actual ventilation rate per occupant and the room air changes resulting from the ventilation rate in each exposure were very different. The purpose of their juxtaposition is to illustrate one circumstance, the ICU exposure, where actual room total outdoor air ventilation was well below recommended levels and where easily achievable increases in ventilation from the actual value are predicted to result in substantial decreases in risk. The exposure conditions result in a data point high up on the vertical limb of the curve, where small changes in ventilation result in large changes in risk. In contrast, in the office building,

Figure 6. Probability of infection as a function of ventilation. These curves are the result of modeling actual exposures, using the Wells-Riley mass-balance equation. In the intensive care (ICU), baseline ventilation was poor, and modest increases would result in marked reductions in transmission. In the office building, baseline ventilation was much better, and major increases would still leave many occupants unprotected.



SOURCE: Nardell EA, Keegan J, Cheney SA, Etkind SC. Airborne infection. theoretical limits of protection achievable by building ventilation. Am Rev Respir Dis 1991;144:302-6.

exposure ventilation was only slightly below national standards. Achievable increases are relatively modest and, therefore, result in a smaller decrease in risk. In this case, the actual data point is near the bottom of the vertical limb of the curve, where each additional infection averted requires a larger and larger increase in ventilation.

A simple way to think of the relationship between risk of infection and ventilation is that each doubling of ventilation reduces the *remaining* risk by approximately half. The ICU exposure was brief (2.5 hrs during a bronchoscopy and intubation), but intensive (i.e., *q* estimated at 250 infectious doses generated per hour), during which 10 of 13 (80%) exposed individuals were infected. Ventilation was so low (150 cfm) that it would be realistic to double it, and even double that value again and again, resulting in protection of almost all of the exposed susceptible occupants. However, the office building exposure was longer (30 days), but much less intensive (*q* estimated at 13 infectious doses generated per hour), resulting in infection of 27 of 67 (40%) exposed workers. In this case, increasing ventilation from the existing 15 cfm outdoor air per occupant (1450 cfm) to the currently recommended 20 cfm would be possible, but is predicted to protect only a few of the 27 workers infected. Even doubling ventilation to 2900 cfm (30 cfm per occupant—highly unusual for an office building—would have protected only about half of those infected, according to the equation.

APPENDIX 2. MICROORGANISM SUSCEPTIBILITY TO UVGI EXPOSURE

The fractional survival of microorganisms exposed to UVGI is expressed in the relationship shown in the equation below. While this equation gives a straight line in semilogarithmic representation, many microorganisms show deviations at the end, e.g., tailing.

$$\frac{N_s}{N_o} = 10^{-K \bullet H_0}$$

where

N_o = number of bacteria exposed
N_s = number of bacteria surviving after an exposure to UVGI
H_0 = fluence, J/m^2
K = decay rate constant (microbe susceptibility factor), m^2/J

This relationship was used to develop Table 1.

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