

Modeling Immune Building Systems for Bioterrorism Defense

Wladyslaw Kowalski¹; William Bahnfleth²; and Amy Musser³

Abstract: This paper presents the results of research on the performance of air-cleaning and air-disinfection systems used for protecting buildings against intentional releases of biological agents. The air-cleaning technologies addressed include dilution ventilation, filtration, and ultraviolet germicidal irradiation. A 40-story commercial office building is modeled using typical occupancy levels and leakage rates for doors, walls, and floors. A steady-state single-zone model is used to predict steady-state conditions resulting from the use of various levels of air cleaning. A transient single-zone model is used to predict transient indoor concentrations from which inhaled doses and estimated casualties are predicted. A transient multizone model is used to evaluate contaminant dispersion and estimate potential casualties. Three design-basis attack scenarios are simulated using various biological weapon agents. Predicted casualties are estimated using an epidemiological model of the dose response curves for each of the agents. The effect of increasing levels of air cleaning is examined under the attack scenarios to evaluate their effectiveness. Results indicate that high levels of protection are possible for building occupants with moderate and affordable levels of air cleaning when filtration is combined with ultraviolet germicidal irradiation. Results also suggest that diminishing returns occur when increased levels of air cleaning are applied. It is hypothesized that the maximum useful size of any air-cleaning system is defined and limited by building physical characteristics alone.

DOI: 10.1061/(ASCE)1076-0431(2003)9:2(86)

CE Database subject headings: Terrorism; Disinfection; Ventilation; Air pollution; Simulation models; Filtration.

Introduction

The anthrax mailing incidents following the terrorist attacks of September 11, 2001, have generated increased interest in the design and application of engineered systems for the protection of buildings against the intentional release of biological agents (USACE 2001). The engineered systems used to protect building occupants against biological and chemical hazards, whether they occur naturally or otherwise, are collectively known as immune-building technologies. Dilution ventilation, filtration, and ultraviolet germicidal irradiation (UVGI) are among the only practical and cost-effective technologies available today that are sufficiently well-understood that can be accurately sized for air-cleaning applications. The performance of these technologies is investigated here to determine whether they are adequate enough to protect building occupants under simulated bioterrorist attacks on a typical commercial office building.

The various methods available for the analysis of contaminants in building air include single-zone models, multizone mod-

els, and computational fluid dynamics. A simple single-zone model is introduced to demonstrate the effectiveness of air-cleaning technologies in reducing the steady-state indoor concentrations of biological weapon (BW) agents released in indoor and outdoor air. A transient single-zone model is used to perform a time-based simulation of several BW agent attack scenarios on an idealized 40-story building. A transient multizone analysis is performed using public domain software, which is capable of modeling the airflows in any multizone building, including infiltration, exfiltration, stack effects in stairwells and elevator shafts, and internal leakage through walls, doors, and windows (Dols et al. 2000). The latter two models are used to compute transient airborne concentrations and inhaled doses of BW agents. Fatality estimates based on the inhaled doses allow comparison of the effectiveness of the air-cleaning technologies.

Air Cleaning Technologies and Design Basis Biological Weapon Agents

The three air-cleaning technologies addressed here, dilution ventilation, filtration, and UVGI, can remove BW agents at predictable rates. Dilution ventilation removes all airborne agents at the same rate by replacing contaminated air with uncontaminated air. For commercial buildings with all-air systems, the outside airflow is typically 15–25% of the total airflow rate. Buildings with airside-economizer systems may, at times, have outside airflow as high as 100% and some ventilation systems may have full-time 100% outside air (OA). In this analysis, the model building has 15% OA.

Removal of agents due to plate-out, or adsorption, on building surfaces is ignored in this study. Adsorption rates depend on particle size, local air velocity, and building materials (Heinsohn 1991)—but these cannot be generalized. Since adsorption re-

¹Research Associate, Indoor Environment Center, Dept. of Architectural Engineering, Pennsylvania State Univ., University Park, PA 16802. E-mail: drkowalski@psu.edu

²Associate Professor and Director, Indoor Environment Center, Dept. of Architectural Engineering, Pennsylvania State Univ., University Park, PA 16802.

³Assistant Professor, Architectural Engineering Program, Univ. of Nebraska, Omaha, NE 68182-0681.

Note. Discussion open until November 1, 2003. Separate discussions must be submitted for individual papers. To extend the closing date by one month, a written request must be filed with the ASCE Managing Editor. The manuscript for this paper was submitted for review and possible publication on August 29, 2002; approved on March 3, 2003. This paper is part of the *Journal of Architectural Engineering*, Vol. 9, No. 2, June 1, 2003. ©ASCE, ISSN 1076-0431/2003/2-86–96/\$18.00.

Table 1. Filtration Rates of Design Basis Biological Weapon Agents

Pathogen	Mean size, μm	Filter Model and Removal Rates, Fraction						
		MERV 6	MERV 7	MERV 8	MERV 10	MERV 13	MERV 15	MERV 16
Influenza	0.098	0.062	0.07	0.11	0.12	0.46	0.71	0.76
Smallpox	0.22	0.037	0.04	0.07	0.08	0.40	0.68	0.71
<i>C. burnetii</i>	0.283	0.037	0.04	0.08	0.08	0.44	0.75	0.77
<i>R. prowazeki</i>	0.283	0.037	0.04	0.08	0.08	0.44	0.75	0.77
<i>L. pneumophila</i>	0.520	0.058	0.08	0.14	0.15	0.69	0.95	0.95
<i>M. tuberculosis</i>	0.637	0.074	0.10	0.18	0.20	0.79	0.98	0.98
<i>C. diphtheria</i>	0.700	0.084	0.12	0.21	0.22	0.83	0.99	0.99
<i>S. pneumoniae</i>	0.707	0.085	0.12	0.21	0.22	0.83	0.99	0.99
<i>R. rickettsii</i>	0.85	0.11	0.15	0.26	0.28	0.90	0.997	0.997
<i>N. asteroides</i>	1.12	0.16	0.22	0.37	0.39	0.96	0.9998	0.9998
<i>Bacillus anthracis</i>	1.12	0.16	0.22	0.37	0.39	0.96	0.9998	0.9998
<i>H. capsulatum</i>	2.24	0.32	0.43	0.67	0.73	0.9997	1.0000	1.0000
Botulinum toxin	2.24	0.35	0.43	0.67	0.73	0.9997	1.0000	1.0000
<i>B. dermatitidis</i>	12.6	0.50	0.60	0.82	0.95	1.0000	1.0000	1.0000

Note: Minimum efficiency reporting value (MERV).

moves contaminants from the air and lowers the indoor airborne concentrations of contaminants, ignoring this factor is conservative. Resuspension of settled particles cannot produce higher airborne concentrations any more than ignoring plate-out effects can. The long-term effects of adsorbed contaminants and associated remediation costs may be an important secondary concern (during cleanup) but are not the subject of the present research.

Filtration is a reliable and predictable means of improving indoor air quality and removing airborne contaminants. The ability of filters to remove microbial contaminants has been demonstrated in a number of laboratory studies (Carpenter et al., 1986; Roelants et al. 1968; Washam 1966; Jensen 1967; Harstad et al. 1967). Models of filter performance have been developed to help predict removal rates for airborne pathogens and allergens (Kowalski et al. 1999). Filtration removes biological contaminants at rates that depend on the particle size distribution of each contaminant. Particles in the bacterial size range have lognormal size distributions and they can be represented adequately by the log-mean diameter of each agent. The filtration rates of these particles have been established with a previously developed filter model and estimates of the logmean diameters (Kowalski et al. 1999; Kowalski and Bahnfleth 2002).

Certain factors may impact the predictions of the filter model used here. Weaponization of BW agents often involves the use of additives that prevent clumping (Kowalski 2002). It is possible that such additives will reduce filtration efficiency, although no information is publicly available on this subject. Prior use of filters will tend to increase the filtration efficiency. Filter bypass can greatly degrade the efficiency of filters depending on the amount of bypass and the recirculation flowrate. Clumping of aerosolized BW agents may increase or decrease filtration rates depending on their size range. The filter model presented here represents ideal conditions (i.e., no bypass, clumping, or additives).

Over 100 potential BW agents exist that may cause fatalities or serious illness, but it is not necessary to evaluate all of them since a limited selection of design basis agents will represent the remainder adequately. Three Class A microbes (1) smallpox virus; (2) anthrax spores; and (3) botulinum toxin have been selected as part of the design-basis BW agents along with 11 others. The three other agents identified by the Centers for Disease Control and Prevention as being Class A agents, *Yersinia pestis* (plague), *Francisella tularensis*, and the hemorrhagic fever viruses (Rotz

et al. 2002) were excluded because insufficient information is available on their UVGI susceptibility.

The three Class A agents and the other 11 BW agents are summarized in Table 1, which lists their mean diameters and the filtration rates for several generic filters.

Botulinum toxin has no specific size but is a powder or a solution in weaponized form. Weaponized toxins are ground to a size of between 1 and 6 μm to facilitate aerosolization. This size range renders them removable by ordinary high-efficiency filters in the minimum efficiency reporting value (MERV) 7–13 range. The mean size of botulinum toxin is assumed to be 2.28 μm .

The mean diameters of the microbes specified in Table 1 are used to compute the mean mass of the microbe by using a density of 1,100 kg/m^3 or 1.1 times the density of water (Bratbak and Dundas 1984). The mean mass of an anthrax spore is computed by this method to be 8.05×10^{-7} μg .

The filtration rates summarized in Table 1 are based on filter performance curves for a limited number of manufacturer's model filters. Since filter performance may vary greatly from manufacturer to manufacturer, these filtration rates are only representative of filter performance. In an actual design situation, the specific manufacturer's filter-performance data should be used to determine filtration rates, unlike Table 1. Furthermore, filters are assumed to be installed with tight seals and without significant filter bypass. Filter bypass degrades overall filter performance by reducing total airflow through the filter. Bypass rates of 10% or higher, which can occur in poor installations, can present a serious problem to any filter's performance—especially at high velocities. The present study is not concerned with the consequences of poor installation and maintenance so filter bypass is not addressed further.

UVGI can be effective against microorganisms when adequately designed and maintained. Methods for modeling and designing UVGI systems have been developed that allow prediction of performance (Kowalski and Bahnfleth 2000). Numerous studies have been performed demonstrating UVGI biocidal effectiveness but only a limited number of airborne microbes have been studied. Data for approximately 12 potential BW agents is used in this study and includes anthrax spores and smallpox (Kowalski et al. 2000). Some studies indicate that UVGI can degrade certain toxins including the botulinum toxin (Shantha and Sreenivasa 1977; Kolesnikova et al. 1983), but there is insufficient data

Table 2. Ultraviolet Germicidal Irradiation Kill Rates of Design Basis Biological Weapon Agents

Pathogen	Rate constant (cm ² /μW-s)	ULTRAVIOLET GERMICIDAL IRRADIATION KILL RATES, FRACTION						
		URV 6	URV 7	URV 8	URV 10	URV 13	URV 15	URV 16
		75 μW/cm ²	100 μW/cm ²	150 μW/cm ²	500 μW/cm ²	2000 μW/cm ²	4000 μW/cm ²	5000 μW/cm ²
Influenza	0.001187	0.044	0.058	0.085	0.257	0.695	0.907	0.949
Smallpox	0.001528	0.056	0.074	0.108	0.318	0.783	0.953	0.978
<i>C. burnetti</i>	0.001535	0.056	0.074	0.109	0.319	0.785	0.954	0.978
<i>R. prowazeki</i>	0.000292	0.011	0.014	0.022	0.070	0.253	0.442	0.518
<i>L. pneumophila</i>	0.002503	0.090	0.118	0.171	0.465	0.918	0.993	0.998
<i>M. tuberculosis</i>	0.002132	0.077	0.101	0.148	0.413	0.881	0.986	0.995
<i>C. diphtheria</i>	0.000701	0.026	0.034	0.051	0.161	0.504	0.754	0.827
<i>S. pneumoniae</i>	0.006161	0.206	0.265	0.370	0.786	0.998	1.0000	1.0000
<i>R. rickettsii</i>	0.000292	0.011	0.014	0.022	0.070	0.253	0.442	0.518
<i>Bacillus anthracis</i>	0.000124	0.005	0.006	0.009	0.031	0.117	0.220	0.267
<i>N. asteroides</i>	0.000123	0.005	0.006	0.009	0.030	0.116	0.218	0.265
<i>H. capsulatum</i>	0.000247	0.009	0.012	0.018	0.060	0.219	0.390	0.461
Botulinum toxin	0	0.000	0.000	0.000	0.000	0.000	0.000	0.000
<i>B. dermatitidis</i>	0.000247	0.009	0.012	0.018	0.060	0.219	0.390	0.461

Note: Ultraviolet germicidal irradiation rating value (URV).

available to predict the removal rates of toxins by UVGI. For the purposes of this study, and to be conservative, UVGI is assumed to have no effect on toxins.

Removal rates, or kill rates, due to UVGI exposure depend on the average intensity of the ultraviolet (UV) field and the species rate constant in air. Kill rates are typically stated in terms of fractions or percentages of removal for each pass through a system. Kill rates can be computed using the single-stage decay equation:

$$KR = 1 - e^{-kt} \quad (1)$$

where KR=kill rate, fraction; k =rate constant, cm²/μW-s I = average intensity, μW/cm²; and t =exposure time, sec.

Eq. (1) is a first-order approximation that is adequate for predictive purposes provided it is used where kill rates are not extremely low or high. Most airborne UVGI rate constants are not known, but data from plate-based (petri dish) and water-based laboratory studies are used where available. Comparisons of published UVGI rate constants for similar species suggest that most plate-based and water-based rate constants are conservative for use in air-based studies (Kowalski et al. 2000). The rate constant for smallpox is unknown but the closely related vaccinia virus is used in its place. Table 2 summarizes the removal rates of the BW agents at various levels of average UVGI intensity. The UVGI rate constants in Table 2 have been summarized from previous laboratory studies and other sources (Kowalski et al. 2000; Knudson 1986; Chick et al. 1963; Mitscherlich and Marth 1984; Sharp 1939; Little 1980; Jensen 1964; Antopol and Ellner 1979; David 1973; Allen et al. 1954; Lidwell and Lowbury 1950; Collier et al. 1955).

The UVGI system sizes in Table 2 are defined with a proposed UVGI rating value (URV) based on the average UV intensity in an enclosure (Kowalski 2002). This rating system parallels the American Society of Heating, Refrigerating, and Air-Conditioning Engineers (ASHRAE) method of rating filters with a MERV and is designed to facilitate matching filters and UVGI systems for controlling biological contaminants. Air velocity is assumed to be at the typical UVGI design velocity of approximately 2.54 m/s (500 fpm), or the same as for most MERV rat-

ings. The exposure time is assumed to be 0.5 s in this model. Dose is the product of intensity and exposure time.

The overall kill rates due to the combination of filtration and UVGI cannot be directly added since the downstream component only operates on the surviving population of the upstream component. The overall kill rate, or removal rate (for filters), can be computed by the following equation:

$$KR_T = 1 - (1 - KR_1)(1 - KR_2) \quad (2)$$

where KR_T =total kill rate, fraction, KR_1 =kill rate or removal rate of Component 1 (i.e., filter), fraction, and KR_2 =kill rate or removal rate of Component 2 (i.e., UVGI), fraction.

Eq. (2) can be expanded indefinitely to account for any number of components. Using this equation, the total kill rates for the combined filters and UVGI systems in Table 1 and Table 2 can be computed and are summarized in Table 3. These values were used to define the characteristics of a combined filter/UVGI system (a MERV/URV system) in the multizone modeling analysis.

Model Building

This study demonstrates the modeling method for an idealized office building with typical parameters and relatively uniform air distribution in which the occupant density is constant throughout the building. The model building represents a 40-story commercial office. The OA ventilation rate, floor area, and occupant densities were based on ASHRAE Standard 62 (ASHRAE 2001). All of the relevant design information for this model building is summarized in Table 4. A single ventilation air-handling unit (AHU) distributes air evenly to all floors. This system was selected mainly for simplicity. In an actual building of this size, it is very likely that there would be multiple air-distribution zones. The system chosen provides the simplest illustration of the methodology presented despite its somewhat improbable size.

Fig. 1 shows the layout of a typical model building floor as drawn using the multizone modeling software. Each floor is divided into five zones with a stairwell and an elevator shaft that runs through all 40 floors. The markers on the walls indicate

Table 3. Combined Removal Rates for Biological Weapon Agents

Pathogen	FILTRATION AND ULTRAVIOLET GERMICIDAL IRRADIATION REMOVAL RATES, FRACTION						
	MERV 6 URV 6	MERV 7 URV 7	MERV 8 URV 8	MERV 10 URV 10	MERV 13 URV 13	MERV 15 URV 15	MERV 16 URV 16
Influenza	0.10	0.12	0.19	0.35	0.84	0.97	0.988
Smallpox	0.09	0.11	0.17	0.37	0.87	0.98	0.994
<i>C. burnetti</i>	0.09	0.12	0.18	0.38	0.88	0.99	0.995
<i>R. prowazeki</i>	0.05	0.06	0.10	0.15	0.58	0.86	0.888
<i>L. pneumophila</i>	0.14	0.19	0.29	0.55	0.97	0.9996	0.9999
<i>M. tuberculosis</i>	0.15	0.19	0.30	0.53	0.97	0.9997	0.9999
<i>C. diphtheria</i>	0.11	0.15	0.25	0.35	0.91	0.997	0.998
<i>S. pneumoniae</i>	0.27	0.35	0.50	0.83	0.9996	1.0000	1.0000
<i>R. rickettsii</i>	0.12	0.16	0.28	0.33	0.92	0.998	0.999
<i>N. asteroides</i>	0.16	0.22	0.37	0.41	0.97	0.9998	0.9999
<i>Bacillus anthracis</i>	0.16	0.22	0.37	0.41	0.97	0.9998	0.9999
<i>H. capsulatum</i>	0.33	0.44	0.67	0.74	0.9997	1.0000	1.0000
Botulinum toxin	0.35	0.43	0.67	0.73	0.9997	1.0000	1.0000
<i>B. dermatitidis</i>	0.50	0.61	0.83	0.95	1.0000	1.0000	1.0000

Note: Minimum efficiency reporting value (MERV); ultraviolet germicidal irradiation rating value (URV).

leakage paths and doorways. Envelope leakage rates were assumed to be 1.5 cm²/m² of surface area with loss coefficients of 0.6, based on an analysis of commercial building leakage studies (Persily 1999). The interior surfaces were assumed to be twice as leaky. Contaminant distribution could be greatly influenced by stack effects, depending on conditions and the release scenario, but this matter is left for future research. Stack effects were avoided in this analysis by keeping indoor and outdoor temperatures similar.

Modeling Inhalation Doses

Data on the actual lethal doses of BW agents are too limited and contradictory to provide accurate estimates of fatalities or infections. However, it is not the intent of this paper to determine actual fatalities but to compare the relative effectiveness of the subject of air-cleaning technologies in terms of reduction in fatalities. A simplified method of dose estimation described as follows proves adequate for this purpose, although no claims are made regarding the method's ability to predict actual fatalities.

Furthermore, the dosages used for the design-basis agents (i.e., the quantities released in the model building) are arbitrary since they simply establish a baseline condition from which to compare equipment performance. A change in the target dose values will affect the quantity of agent released in the model but will not affect the comparative performance of the air-cleaning equipment.

Each BW agent possesses a mean lethal dose (LD₅₀), which is the number of microbes or micrograms (for toxins) inhaled that will cause 50% fatalities in a population, and a mean infectious dose (ID₅₀) that will cause a similar percentage of infections. For the purposes of this study, the term casualties will be used to refer to fatalities and the ID₅₀ will not be used here although it could be substituted for the LD₅₀ in this analysis if desired. In order to predict fatalities produced by exposure to levels other than the LD₅₀, it is necessary to use an epidemiological model. The distribution y of fractional new fatalities in a population that receives a dose x follows a Gaussian (normal) distribution curve centered around the LD₅₀, i.e.,

$$y = \frac{1}{\sigma\sqrt{2\pi}} e^{-0.5(x-\mu)^2/\sigma^2} \quad (3)$$

Table 4. Building Model Input Data

Outside air percentage	15%
Total airflow	53,464 m ³ /min (1,888,081 cfm)
Outside airflow	8,020 m ³ /min (283,212 cfm)
Outside air per person	0.57 m ³ /min (20 cfm)
Return airflow	45,444 m ³ /min (1,604,869 cfm)
Air change rate	1.60 h ⁻¹
Floor area, each floor	2,500 m ² (26,909 ft ²)
Floor height	3 m (9.84 ft)
Number of floors	40
Total building volume	300,000 m ³ (10,594,014 ft ³)
Total floor volume	7,500 m ³ (264,850 ft ³)
Outside airflow/sq.ft.	37.76 m ³ /min (1.07 cfm)
Occupancy	14,160 persons
Indoor air temperature	20 C (68 F)
Outdoor air temperature	23 C (73.4 F)

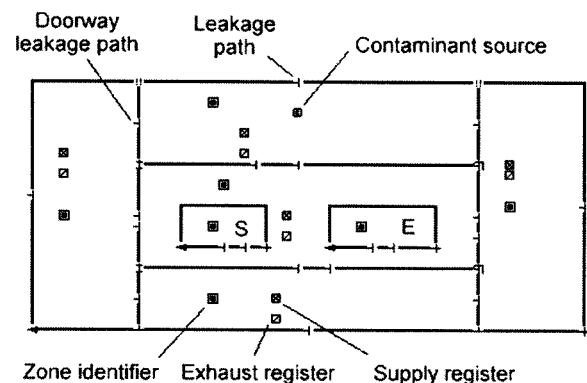


Fig. 1. Floor plan for multizone building model five zones shown for each floor with stairwell (S) and elevator shaft (E) connecting all 40 floors

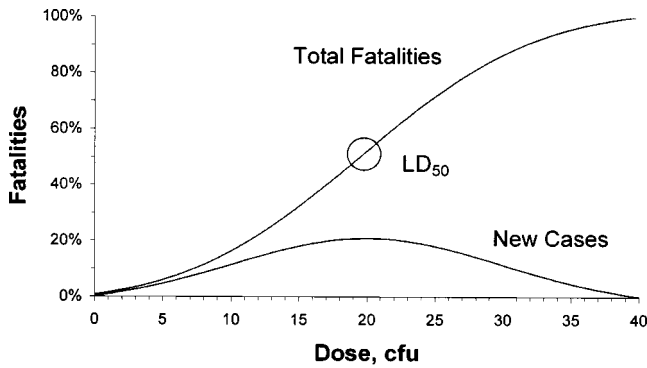


Fig. 2. Epidemiological curves for infectious agent with LD_{50} of 20 cfu

where y = new fatalities, fraction of total population; σ = standard deviation of dose, cfu for pathogens or μg for toxins; μ = mean lethal dose, cfu for pathogens or μg for toxins; and x = dose, cfu for pathogens or μg for toxins.

Eq. (3) produces a normal curve representing the new fatality rate as shown by the lower new cases curve in Fig. 2. The new fatalities are the additional fatalities that occur at each discrete dose increment. Integrating the new fatalities rate over the dose from zero to x generates the upper total fatalities curve in Fig. 2.

For any given dose inhaled by the occupants of a building, it is necessary to predict the total casualties or infections. It would be most convenient to compute these directly for any specified dose, but Eq. (3) cannot be integrated in closed form. Other classic epidemiological models (i.e., Daley and Gani 1999; Frauenthal 1980) are not easily converted into functions of the LD_{50} , which is what is needed to define a generic casualty curve that can be applied to any BW agent. To serve the needs of those who may not care to perform numerical integration of Eq. (3), the writers have developed an approximate closed-form solution by modeling the total fatalities curve in Fig. 2 as a Gompertz curve (Bailey 1975). The Gompertz curve defines a variable y in terms of a function of the variable z as follows:

$$y = a^{b^z} \quad (4)$$

where a = a fractional constant, typically 0.01–0.5; b = a fractional constant, typically 0.01–0.5; and z = a variable.

Eq. (4) will approximate the total fatalities in Fig. 2 with reasonable accuracy if appropriate values for the constants a and b are found. Eq. (4) must be scaled so that it satisfies the constraint $y = 0.5$ at the LD_{50} value. This condition is met when $a = 0.5$ and the exponent z is defined so that $z(LD_{50}) = 0$. The resulting equation for y , which represents the fractional percentage of fatalities as a function of an arbitrary dose for a given LD_{50} value is

$$y = 0.5^{b^{(x-LD_{50})/LD_{50}}} \quad (5)$$

The constant b was estimated by iteration and satisfactory results obtained if it is approximately equal to 0.1. Therefore, the final closed form of the equation for prediction of total fatalities is as follows:

$$y = 0.5^{0.1^{(x-LD_{50})/LD_{50}}} \quad (6)$$

Eq. (6) is not intended to be used for epidemiological purposes, which the classic epidemiological model serves, but is intended to be used for conveniently estimating fatalities for the purpose of comparing the performance of air-cleaning systems in building-attack simulations. Although Eq. (6) will tend to deviate from the

results of Eq. (3) under extremes of high or low doses, Eq. (6) should produce a reasonable estimate of fatalities in accordance with the general form of classical epidemiological models and this should be sufficient to provide a relative comparison of the effectiveness of air-cleaning systems in the model building.

Other epidemiological models are available, including both empirical and theoretical models (Wells 1955; Haas et al. 1999; Nicas 1996; Haas 2002), but none of these lend themselves well to analytical use and none of the empirical data available from these sources invalidates the simplified model of Eq. (6). It must be reiterated that Eq. (6) is not intended for use in making explicit epidemiological predictions but is strictly for the purpose of comparing air-cleaning equipment performance. To that end, it serves as well as any of the more complex and unwieldy epidemiological models would if they could be adapted.

Given any airborne concentration of a BW agent, the breathing rate can be used to compute the inhaled dose. The efficiency with which particles are absorbed by the lungs varies from about 10–100% depending on particle size and activity level (Heinsohn 1991). Instead of evaluating each BW agent, it is assumed in this study that the absorption efficiency is 90% for all agents. BW agents smaller than about $10 \mu\text{m}$ will be absorbed at a rate of 90% or less and agents in the $1 \mu\text{m}$ range may only be absorbed at about 30%—so the assumption is conservative (Heinsohn 1991).

Breathing rates vary with level of activity but for the purposes of this analysis, it is conservatively assumed that the activity level is moderate and the breathing rate of all occupants is $0.02 \text{ m}^3/\text{min}$ ($0.71 \text{ ft}^3/\text{min}$) based on Heinsohn (1991). The dose absorbed by an occupant is then defined by the following equation:

$$D = \eta B_r C_a E_t \quad (7)$$

where D = absorbed dose (cfu for microbes or μg for toxins); η = absorption efficiency of lungs (assumed to be 90%); B_r = breathing rate, m^3/min (ft^3/min); C_a = airborne concentration, cfu/m^3 or mg/m^3 (cfu/ft^3); and E_t = exposure time, s.

The exact LD_{50} for the BW agents studied here are assumed or used per values stated in the references. However, since the physical quantity of each agent released into the building is based on the LD_{50} , the actual values used are not of critical importance. If a more appropriate LD_{50} becomes known, the results of this analysis can simply be scaled to the new LD_{50} value without redoing the analysis. For this analysis, the quantity of each agent released into the building is that which would cause 99% predicted fatalities—or what could be called an LD_{99} . The exact value of the LD_{99} and the quantity of each BW agent it represents is irrelevant because the dose modeling is generic and will produce the same total fatalities curve as in Fig. 2, but with a different axis of values that depends on building characteristics (i.e., airflow, volume, leakage rates, plate-out effects, etc.). In fact, it is not only unnecessary but it is imprudent to publish the quantities of agents used in these simulations. The only factors that are actually important are the physical characteristics of the microbes since these will determine the removal rates by the air-cleaning systems.

Modeling Attack Scenarios

Table 5 summarizes the five scenarios and the three different modeling tools used to model them. The scenarios simulate different types of outdoor and indoor releases and these are described in more detail in the following paragraphs.

Table 5. Attack Scenarios and Modeling Tools

Model	Description	Release location	Analytical method
SSi	Steady state single zone	Indoor	System model equation
SSo	Steady state single zone	Outdoor air	System model equation
Scenario A	Transient single zone	Outside air intakes	Transient computational model
Scenario B	Transient single zone	Air-handling unit	Transient computational model
Scenario C	Transient multizone	General area	CONTAMW program

The first two attack scenarios demonstrate a simple steady-state system model. Since this model can only predict the final steady-state concentrations of a release event, it cannot be used to accurately estimate the inhaled dose or casualties. Scenario A, a sudden BW agent release in the OA intakes, and Scenario B, a continuous release inside the AHU, were modeled using a single-zone model of the building. Because the building is treated as a single zone in this analysis, all zones have the same airborne concentration distributions. The inhalation doses are estimated and predicted casualties computed for these cases.

The final case in Table 5 is Scenario C, in which the BW agent is released on one floor of the 40-story building. This analysis requires use of the multizone modeling program since different floors of the building will experience different airborne concentrations over time. This model includes the effects of internal wall and floor leakage, which is not relevant to the single-zone analyses.

Steady-State Single-Zone Model

The steady-state single-zone model, also called a system model, is an algebraic equation that calculates steady-state conditions for any constant source of contaminants in indoor or outdoor air at constant flowrates. The system model in Eq. (8) has been adapted from Grimm and Rosaler (1990) with minor changes to the nomenclature and units in order to facilitate analysis of airborne microorganisms and their removal rates by filtration and UVGI

$$C_i = \frac{G_i + (P_i Q_i + P_f P_u Q_m) C_0}{Q_x + K_d A_d + Q_r (1 - P_f P_u)} \quad (8)$$

where C_i = steady-state indoor concentration, cfu/m³; G_i = indoor generation or release rate, cfu/min; P_i = penetration from outdoors through cracks, fraction; Q_i = air leakage from cracks, m³/min; P_f = penetration through filter, fraction; P_u = penetration (survival) through UVGI system, fraction; Q_m = OA flowrate through filter, m³/min; C_0 = outdoor concentration, cfu/m³; Q_x = exhaust air, m³/min; K_d = indoor deposition velocity, m/min; A_d = indoor deposition area, m²; and Q_r = return airflow, m³/min.

In Eq. (8), the units of cfu may be used to represent bacteria, fungal spores, or viruses even though viruses are more properly defined by plaque-forming units (PFU).

Table 6 summarizes the input data for two cases—an indoor release of smallpox and an outdoor concentration of anthrax spores. The airflows represent a single floor of the 40-story building. As noted previously, plate-out is ignored ($A_d = 0$), which is a conservative assumption that increases indoor contaminant concentrations. The air leakage, Q_i , is assumed to be 1% of the total return airflow. The filter removal rates and UVGI kill rates are the same as those previously presented in Tables 1 and 2, but three additional MERV/URV ratings (10, 11, and 12) are included in this example and these values (only) are shown in Table 6.

Fig. 3 summarizes the results of the two release scenarios side-by-side. The steady-state concentrations reach minimum values at levels of air cleaning that correspond to a MERV 13 filter combined with an URV 13 UVGI system. Further increases in the level of filtration or the UVGI intensity will not significantly decrease indoor concentrations. This is an important aspect of air-cleaning component performance to note—system effects limit the overall removal efficiencies they can achieve.

The steady-state results for the previous examples are not directly useful since it is necessary to calculate inhaled dosages of the agents that would occur over time and then use these dosages to estimate casualties. These topics are now addressed prior to performing the detailed transient attack simulations.

Table 6. Summary of Systems Model Input Data

Event statistics	Value
Total airflow	1,336 m ³ /min
Return airflow	1,136 m ³ /min
Makeup air	200 m ³ /min
Leakage	11 m ³ /min
Ultraviolet exposure time	0.55
(a) Biological weapon agent: Smallpox virus	
Release rate	1,000 cfu/min
Outdoor concentration	N/A
MERV/URV rating	
Filter removal	
10/10	7.9%
11/11	14%
12/12	23%
UVGI/Kill rate	
10/10	32%
11/11	53%
12/12	68%
(b) Biological weapon agent: Anthrax spores	
Release rate	N/A
Outdoor concentration	1,000 cfu/min
MERV/URV rating	
Filter removal	
10/10	0.8%
11/11	1.5%
12/12	2.3%
UVGI/Kill rate	
10/10	39%
11/11	57%
12/12	76%

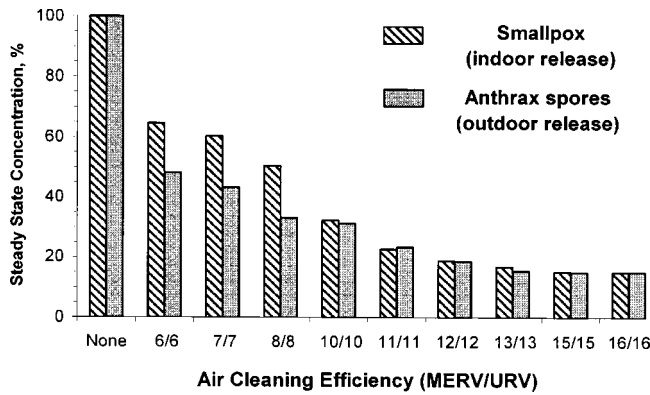


Fig. 3. Steady-state results for indoor releases and outdoor concentrations at various levels of air-cleaning efficiency (MERV/URV rating)

Transient Modeling and Casualty Prediction

The three BW agent release scenarios considered for modeling were summarized in Table 5. Additional release scenarios are possible, but the ones summarized here represent the most commonly cited attack scenarios and will produce representative results. For example, if the release occurred suddenly in the supply duct or AHU, the effect would be essentially identical to the sudden release in the OA intakes (minus the filtering effect). If a sudden release occurred in a general area (i.e., the main floor or first-floor lobby) the occupant exposure would be less severe than a gradual release in a general area. These scenarios may not represent the worst cases for every possible type of building, ventilation system, or operating condition, but they will be basically representative of the most likely or threatening attack scenarios in typical commercial office buildings with constant-volume air systems.

Releases of BW agents would likely occur covertly and no one would initially be aware that a release was occurring. In such a situation, the released agents might be inhaled for the duration of an 8 h day. The gradual release of a BW agent inside the AHU may cause the maximum number of casualties, depending on whether there are air-cleaning devices and where they are placed. In the baseline condition, the building is assumed to have no air-cleaning capacity other than that due to dilution ventilation.

A sudden release in the OA intakes might result from dumping a powder or liquid into the air intakes or by the use of an explosive device. Fig. 4 illustrates this type of release scenario. The release of an agent like anthrax in the OA intakes of a large office building has been suggested as one type of attack that would place the perpetrator at little risk and cause high casualties. Since many buildings have ground-level OA intakes that are open and unprotected, this is a plausible scenario. This scenario also encompasses the release of an agent in the OA if the released cloud drifted to the OA intakes.

The effect of a slow release of a BW agent in the AHU would bear a resemblance to the previous figure and no additional diagram is provided for Scenario B. The slow release of a BW agent in the general areas could be accomplished through the use of concealed aerosolization devices. The contaminants would spread rapidly throughout the building if the air-distribution system recirculated return air, but the highest concentration would remain the immediate area of the release. Fig. 5 illustrates Scenario C for a building with a recirculating system.

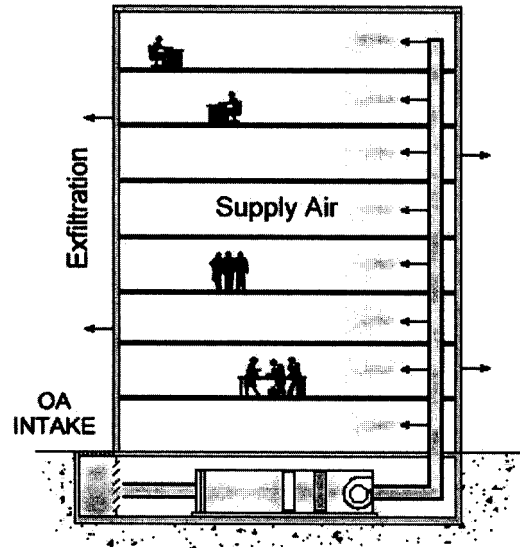


Fig. 4. Scenario A—sudden release of biological weapon agent inside outside air intake

In order to establish a design-basis scenario, it is necessary to know the quantity of agent that is released. This quantity cannot be arbitrarily chosen since too small or too large a quantity will distort the results of an evaluation. It is assumed, therefore, that the quantity of agent released is that which is sufficient to cause 99% predicted fatalities when released continuously in the AHU without any air-cleaning components other than dilution. This is Scenario A, and the same quantities used in this scenario are used for the other two scenarios so as to maintain a fair comparative basis for evaluating the results. The reason 99% is used instead of 100% is that the epidemiological curves approach 100% asymptotically (Fig. 2) and using 100% exactly will exaggerate the quantities needed. The actual quantity that will cause 99% fatali-

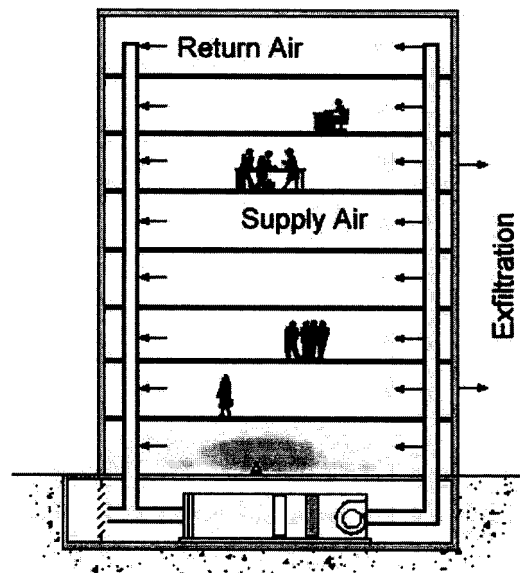


Fig. 5. Scenario C—slow release of biological weapon agent in general area with aerosolization device

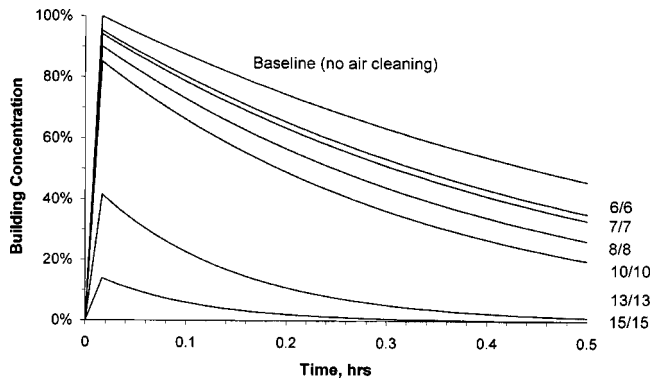


Fig. 6. Concentrations for sudden release in outside air intakes—baseline condition compared with six levels of air cleaning (identified by MERV/URV ratings)

ties cannot be known in advance. It is necessary to run iterative analysis beginning with an assumed quantity until 99% fatalities are achieved.

Scenario A, the sudden release in the OA intakes, will distribute the agent to each zone in proportion to the supply airflow rate. In the cases where filters are operating, it is necessary to account for the filtration rate of the first pass through the filter by reducing the initial contaminant source by the removal rate.

Scenario C, the gradual release in a general area, was modeled by placing the contaminant source in a first-floor zone. This scenario requires the use of a multizone modeling program. Realistic values for wall and door leakage were used in this model, as opposed to the two previous models in which no internal leakage was modeled since it would have had no effect in a single zone.

Results and Discussion

The simulations of the three main attack scenarios presented here are based on operating conditions that include a baseline condition in which no filters or UVGI systems are present and six levels of air cleaning indicated by MERV/URV ratings. A total of 63 analyses were run and the results are discussed in the following sections for each of the three attack scenarios.

Scenario A—Sudden Release in OA Intake

In this scenario, the BW agent is released suddenly in the OA intakes. This causes a spike in the indoor airborne concentrations that drops off over the next 2 h as the agent is purged or removed by the air-cleaning components. Fig. 6 shows the concentrations in the model building for the first hour for six of the seven air-cleaning efficiency levels (MERV/URV).

The diminishing returns effect appears again in this scenario as shown in Fig. 7. The predicted casualties converge to zero with hardly any significant difference (only approximately 3%) between the MERV/URV 11/11 and 13/13 systems. Once the predicted fatalities are reduced to approximately zero, further increases in air-cleaning efficiency (MERV/URV) can produce little additional benefit.

Scenario B—Gradual Release in Air Handling Unit

The gradual release of a BW agent inside the AHU causes indoor concentrations to increase throughout the building until the

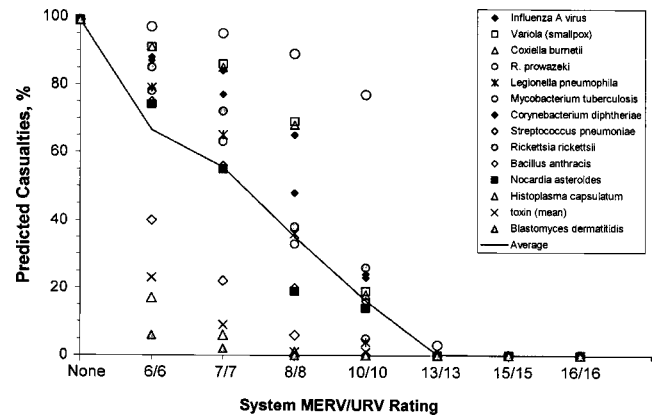


Fig. 7. Diminishing returns effect of increased air cleaning (MERV/URV) for Scenario A—line indicates average for 14 biological weapon agents

steady-state condition is approached. Steady state is achieved in about 2 h when the OA flowrate is 15%. During the 8 h period when occupants are exposed to the agent, they inhale a quantity that depends on their breathing rate. Occupants will have acquired lethal doses of the agent at different times, depending on their susceptibility and breathing rate. It is assumed that the occupancy is continuous for this and the other scenarios. This is conservative since large buildings will tend to have some transient occupants who will not receive a full dose.

The six air-cleaning configurations that were previously described produce a range of reductions in predicted fatalities, as shown in Fig. 8. This figure summarizes the results for a smallpox release in the model building. The results produce an epidemiological curve that shifts to the right as the airborne concentrations decrease, as would be expected.

All BW agents produce similar results, depending on the rate at which they are removed (Table 4). The predicted fatalities can be reduced by filters in the MERV 11–13 range combined with UVGI in the URV 11–13 range, but increasing levels of air cleaning beyond this brings no significant further decrease in occupant casualties. Fig. 9 illustrates this diminishing returns effect for Scenario B showing the results for 14 BW agents.

Diminishing returns are observable in Fig. 9, but unlike the previous scenario, the predicted fatalities converge not on zero, but on a minimum value, which in this case is approximately 9%. The diminishing returns effect is clearly a function of the building

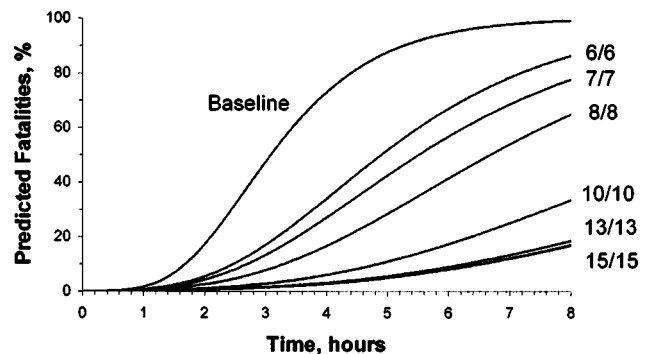


Fig. 8. Predicted fatalities for smallpox release under Scenario B, gradual release in air-handling unit—MERV/URV ratings shown at right

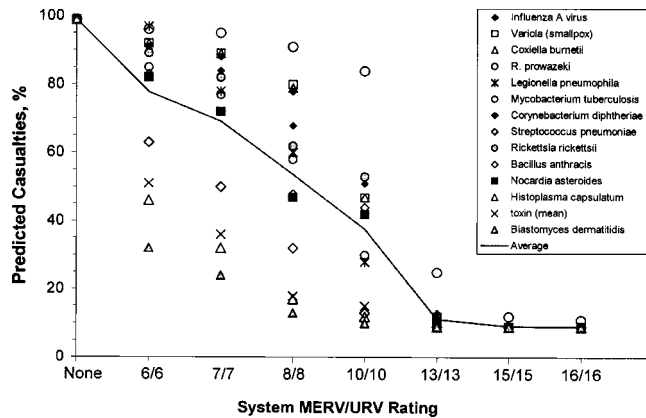


Fig. 9. Diminishing returns from increasing air cleaning for Scenario B—line shows averages of predicted fatalities for 14 biological weapon agents

volume and ventilation system flow rate, which are defining characteristics of each individual building.

Scenario C—Gradual Release on Main Floor

This release occurs gradually and covertly on the first floor with no releases on other floors. Due to the copious input requirements and output data generated by the transient multizone simulation, only the three Class A BW agents are evaluated—anthrax spores, smallpox, and botulinum. These agents are fairly well-representative of the full array of BW agents as may be observed by a review of Tables 1–3.

Predicted concentrations for the case of an anthrax release are shown in Fig. 10 for the baseline condition (no air cleaning). It can be observed that the airborne concentration on the main floor rapidly reaches steady state, while the concentrations on the remaining floors only reach a fraction of the main-floor level. In the absence of interfloor transfers driven by stack effect, the difference in concentration between the second floor and the remaining floors is not significant.

Fig. 11 shows the predicted fatalities, as a percentage of occupants, for the main floor versus the other floors in the 40-story building model. The differences in the concentrations between the other floors are relatively insignificant, as would be expected with the forced ventilation system, and the curves all overlap. In this simulation, the same number of occupants is on the main floor as

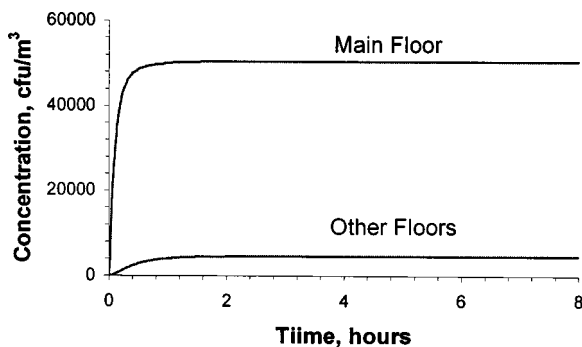


Fig. 10. Airborne concentrations of anthrax on floor with release (main floor) compared with other floors for baseline condition (no air cleaning)

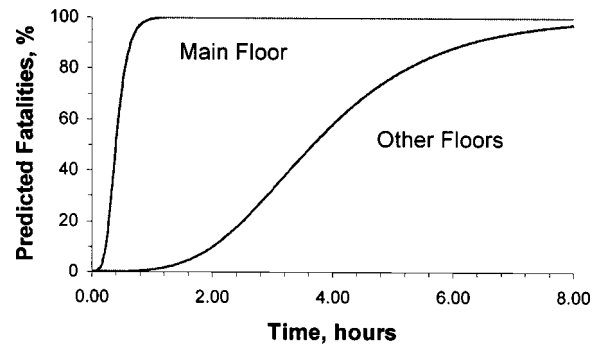


Fig. 11. Predicted fatalities from anthrax release on main floor compared with other floors for baseline condition (no air cleaning)

on each of the other floors. Obviously, the presence of a larger number of occupants on the floor with the release would increase the casualties. This might be the case when the main floor has heavier traffic from the other floors, but estimating transient occupancy levels requires empirical data and so is a matter left for future research and actual applications of the model.

The results of this multizone analysis can be impacted by stack-effect pressurization when outdoor temperatures fall and differ significantly from indoor temperatures. Imbalanced pressurization by mechanical systems could also cause unintended spread of contaminants between floors. Both of these effects alter the manner in which agents are distributed throughout a building, and would produce more variation in concentration on the nonrelease floors than is seen here. These effects are not dealt with here due to their complexity, but they have been analyzed elsewhere (Musser et al. 2002). Results of that analysis suggest that airborne concentrations can be increased as much as 20% in zones remote from the source.

Simulated releases of other BW agents produced results similar to those of the anthrax release shown in Fig. 11. Fig. 12 shows the diminishing returns effect for releases of anthrax, smallpox, and botulinum in a general area. The result is the same type of curve seen in all the previous analyses, including the steady-state system model, and diminishing returns dictate the limiting air-cleaner efficiency (MERV/URV). Other than differences in the final limiting condition, this characteristic curve is not affected by the differences between the scenarios or by the specific BW agent released. This suggests that there exists a limiting system size for any building.

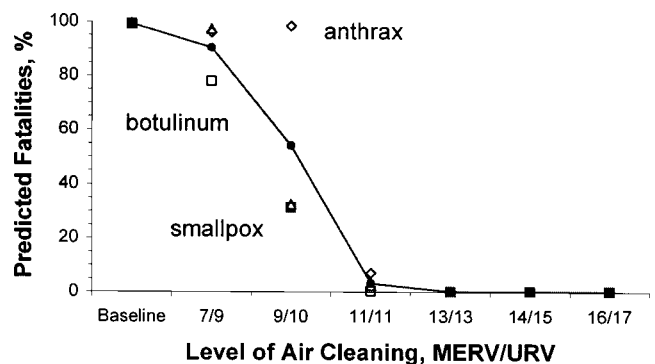


Fig. 12. Diminishing returns effect for Scenario C—line shows average from three design basis biological weapon agents

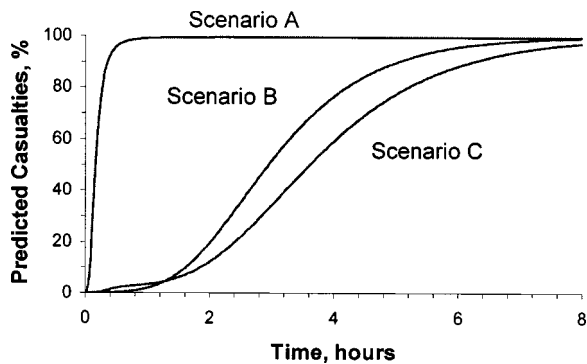


Fig. 13. Comparison of predicted casualties for all three release scenarios when no air-cleaning components are present

Finally, it is of some interest to compare the three design-basis scenarios to determine the relative severity of each. Fig. 13 shows a comparison of the predicted casualties for the three scenarios for the baseline condition, in which no filtration or UVGI is present. In all three of these scenarios, the same quantity of agent is released. These curves represent the percent of casualties that would be produced during the indicated time periods. Although Scenario A, the sudden release in the OA intakes, produces more rapid casualties, it converges to the same fatality rate as Scenario B. Since these fatalities would actually occur several hours (for toxins) or several days (for pathogens) after the attack, it is a matter of debate whether Scenario C is worse than Scenario A. Both scenarios are seen to be more severe than Scenario C—the gradual release in a general area.

The previous comparison may not be entirely realistic, however, since some level of filtration (i.e., prefilters or dust filters) will usually be present in most buildings. The results shown here apply strictly to large commercial office buildings and may not apply to facilities with drastically different operating parameters such as auditoriums and stadiums.

Conclusions

A modeling method that uses public-domain multizone-network airflow-modeling software to evaluate the performance of air-cleaning components for protecting occupants against the release of biological weapon agents has been described and its use illustrated. These methods can be adapted and applied to many types of buildings and facilities. The method is applicable mainly to buildings with mixing ventilation in conditioned spaces. It is less appropriate for analyzing stratified spaces in which the details of room airflow would significantly influence exposure by creating highly nonuniform contaminant-concentration distributions.

The results presented here suggest that the benefits of incremental increases in the performance of air-cleaning components installed in a central recirculating air-distribution system diminish to a point at which further increase has negligible benefit. For the model 40-story commercial office building with 15% OA studied here, filtration in the range of approximately MERV 13–15, combined with a UVGI dose of approximately $1,000 \mu\text{W}\cdot\text{s}/\text{cm}^2$ (URV 13–15 for 0.5 s) represent a limiting condition beyond which no significant improvement in the level of protection occurs. This result is promising since these levels of air cleaning are affordable for most commercial buildings. It is important to note that changing the ventilation rate alters the point of diminishing returns. For

a lower ventilation rate, a higher air-cleaning efficiency might be warranted and a system with a higher ventilation rate would benefit less from very high air-cleaning efficiency. Dilution, filtration, and UVGI all contribute to the removal of BWs and any of them may be the limiting factor in a particular system.

Further research is necessary to determine whether these conclusions apply generally to other types of buildings and under other conditions of operation. Research remains to be done on the effects of varying total airflow volume, the effects of variable air volume systems on contaminant dispersion, stack effects, imbalanced airflow-system operation, and the effects of building characteristics such as internal and external leakage and plate-out. Also worth studying is the effect of air-cleaning systems on the reduction of naturally occurring airborne diseases and allergies, but this is a matter that requires empirical data from actual applications. Many other related areas of biodefense technology require further research but the application of air-cleaning technologies appears to be both feasible and affordable at the present time.

References

- Allen, E. G., Bovarnick, M. R., and Snyder, J. C. (1954). "The effect of irradiation with ultraviolet light on various properties of typhus rickettsiae." *J. Bacteriol.*, 67, 718–723.
- American Society of Heating, Refrigerating, and Air Conditioning Engineers (ASHRAE). (2001). "Ventilation for acceptable indoor air quality." *ASHRAE standard 62-2001* Atlanta.
- Antopol, S. C., and Ellner, P. D. (1979). "Susceptibility of *Legionella pneumophila* to ultraviolet radiation." *Appl. Environ. Microbiol.*, 38(2), 347–348.
- Bailey, N. T. J. (1975). *The mathematical theory of infectious diseases and its applications*, 2nd Ed., Hafner, New York.
- Bratbak, G., and Dundas, I. (1984). "Bacterial dry matter content and biomass estimations." *Appl. Environ. Microbiol.*, 48, 755–757.
- Carpenter, G. A., Smith, W. K., MacLaren, A. P. C., and Spackman, D. (1986). "Effect of internal air filtration on the performance of broilers and the aerial concentrations of dust and bacteria." *Br. Poult. Sci.*, 27, 471–480.
- Chick, E. W., Hudnell, J. A. B., and Sharp, D. G. (1963). "Ultraviolet sensitivity of fungi associated with mycotic keratitis and other mycoses." *Sabouviad*, 2(4), 195–200.
- Collier, L. H., McClean, D., and Vallet, L. (1955). "The antigenicity of ultra-violet irradiated vaccinia virus." *J. Hyg.*, 53(4), 513–534.
- Daley, D. J., and Gani, J. (1999). *Epidemic modelling: An introduction*, Cambridge University Press, New York.
- David, H. L. (1973). "Response of mycobacteria to ultraviolet radiation." *Am. Rev. Respir. Dis.*, 108, 1175–1184.
- Dols, W. S., Walton, G. N., and Denton, K. R. (2000). *CONTAMW 1.0 user manual*, NIST Gaithersburg, Md.
- Frauenthal, J. C. (1980). *Mathematical modeling in epidemiology*, Springer, New York.
- Grimm, N. R., and Rosaler, R. C. (1990). *Handbook of HVAC design*, McGraw-Hill, New York.
- Haas, C. N. (2002). "On the risk of mortality to primates exposed to anthrax spores." *Risk Anal.*, 22(2), 189–193.
- Haas, C. N., Rose, J. B., and Gerba, C. P. (1999). *Quantitative microbial risk assessment*, Wiley, New York.
- Harstad, J. B., Decker, H. M., Buchanan, L. M., and Filler, M. E. (1967). "Air filtration of submicron virus aerosols." *Am. J. Public Health*, 57(12), 2186–2193.
- Heinsohn, R. J. (1991). *Industrial ventilation: Principles and practice*, Wiley, New York.
- Jensen, M. (1967). "Bacteriophage aerosol challenge of installed air contamination control systems." *Appl. Microbiol.*, 15(6), 1447–1449.
- Jensen, M. M. (1964). "Inactivation of airborne viruses by ultraviolet irradiation." *Appl. Microbiol.*, 12(5), 418–420.

- Knudson, G. B. (1986). "Photoreactivation of ultraviolet-irradiated, plasmid-bearing, and plasmid-free strains of *Bacillus anthracis*." *Appl. Environ. Microbiol.*, 52(3), 444–449.
- Kolesnikova, V. A., Shibaeva, I. V., and Ivanov, K. K. (1983). "Effect of photooxidation on biological properties of botulinum neurotoxin A." *Biokhimiia*, 48(1), 33–39.
- Kowalski, W. J. (2002). *Immune building systems technology*, McGraw-Hill, New York.
- Kowalski, W. J., and Bahnfleth, W. P. (2000). "Effective UVGI system design through improved modeling." *ASHRAE Trans.*, 106(2), 4–15.
- Kowalski, W. J., and Bahnfleth, W. P. (2002). "MERV filter models for aerobiological applications." *Air Media, Summer*, 13–17.
- Kowalski, W. J., Bahnfleth, W. P., Whittam, T. S. (1999). "Filtration of airborne microorganisms: Modeling and prediction." *ASHRAE Trans.*, 105(2), 4–17.
- Kowalski, W. J., Bahnfleth, W. P., Witham, D., Severin, B. F., and Whittam, T. S. (2000). "Mathematical modeling of UVGI for air disinfection." *Quant. Microbiol.*, 2(3), 249–270.
- Lidwell, O. M., and Lowbury, E. J. (1950). "The survival of bacteria in dust." *Annu. Rev. Microbiol.*, 14, 38–43.
- Little, J. S., Kishimoto, R. A., and Canonico, P. G. (1980). "In vitro studies of interaction of rickettsia and macrophages: Effect of ultraviolet light on *Coxiella burnetii* inactivation and macrophage enzymes." *Infect. Immun.*, 27(3), 837–841.
- Mitscherlich, E., and Marth, E. H. (1984). *Microbial survival in the environment*, Springer, Berlin.
- Musser, A., Kowalski, W., and Bahnfleth, W. (2002). "Stack and mechanical system effects on dispersion of biological agents in a tall building." *Proc., 9th Symp. on Measurement and Modeling of Environmental Flows, Int. Mechanical Engineering Congress and Exposition (IMECE '02)*, New Orleans, American Society of Mechanical Engineers (ASME), New York, Paper No. IMECE2002-33862.
- Nicas, M. (1996). "An analytical framework for relating dose, risk, and incidence: An application to occupational tuberculosis infection." *Risk Anal.*, 16(4), 527–538.
- Persily, A. K. (1999). "Myths about building envelopes." *ASHRAE J.*, 41(3), 39–47.
- Roelants, P., Boon, B., and Lhoest, W. (1968). "Evaluation of a commercial air filter for removal of viruses from the air." *Appl. Microbiol.*, 16(10), 1465–1467.
- Rotz, L. D., Khan, A. S., Lillibridge, S. R., Ostroff, S. M., and Hughes, J. M. (2002). "Report summary-public health assessment of potential biological terrorism agents." *Emerg. Infect. Dis.*, 8(2), 225–230.
- Shantha, T., and Sreenivasa, M. (1977). "Photo-destruction of aflatoxin in groundnut oil." *Indian J. Technol.*, 15, 453.
- Sharp, G. (1939). "The lethal action of short ultraviolet rays on several common pathogenic bacteria." *J. Bacteriol.*, 37, 447–459.
- United States Army Corps of Engineers (USACE). (2001). "Protecting buildings and their occupants from airborne hazards." *TI 853-01*, Washington, D.C.
- Washam, C. J. (1966). "Evaluation of filters for removal of bacteriophages from air." *Appl. Microbiol.*, 14(4), 497–505.
- Wells, W. F. (1955). *Airborne contagion and air hygiene*, Harvard University Press, Cambridge, Mass.