Ventilation of General Hospital Wards for Mitigating Infection Risks of three kinds of viruses including Middle East Respiratory Syndrome Coronavirus (MERS-*CoV*)

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Abstract

This study investigates the effectiveness of ventilation design strategies for general hospital wards in terms of virus removal capacity. A typical semi-enclosed six-bed general ward of Hong Kong hospitals and three respiratory viruses, namely Middle East Respiratory Syndrome Coronavirus (MERS-*CoV*), Severe Acute Respiratory Syndrome Coronavirus (SARS-*CoV*) and H1N1 influenza virus, were chosen for the computational fluid dynamics (CFD) simulation of airflow field and virus dispersion inside the ward. The results demonstrated that the location of an infected patient would affect the infection risks to other occupants and healthcare workers inside the same hospital ward, and an increased air change rate in the ward could reduce the risk of infection from direct contact and inhalation. It was found that an air change rate of 9 h^{-1} could effectively minimize the deposition and floating time of respiratory virus particles while maximizing energy efficiency. This study should provide a useful source of reference for the hospital management to mitigate the risk of infection with MERS or other airborne transmitted viruses through better ventilation design strategies.

Keywords

Ventilation, virus dispersion, hospital general wards, CFD, Middle East Respiratory Syndrome (MERS)

Introduction

According to a statistical report by the Hong Kong Hospital Authority,[1] the in-patient discharges and deaths were continuously increasing from 2003 to 2013. The 2012/13 overall number of in-patient discharges and deaths was 1,027,005 and that of day-patient was 516,127 in Hong Kong. Among all patients, 33.4% were day patients.

To prevent nosocomial or healthcare-associated infections, especially airborne ones, hospital hygiene and infection control are necessary. The Centres for Disease Control and Prevention (CDC) provides guidance to help healthcare personnel to follow standard, contact, and airborne precautions when caring for hospitalized patients with known or suspected viral infections.[2] Effective prevention measures, e.g. an airborne infection isolation room (AIIR), are especially crucial for control of acute respiratory infectious threats.

Proper ventilation also plays a key role in infection control by minimizing airborne bacteria and viruses. Both the spread of Severe Acute Respiratory Syndrome Coronavirus (SARS-*CoV*) during the largest nosocomial SARS outbreak in Hong Kong and the recent outbreak of Middle East Respiratory Syndrome (MERS) in the South Korean hospitals revealed that airborne disease transmission through inefficient hospital ward ventilation systems can lead to dire health consequences.[3-5] For a balanced ventilation that delivers indoor air quality and energy efficiency, general hospital wards have been designed to meet certain air change requirements.[2,6-10] For instance, an air change rate (*ach*) ranging from 2 to 6 h^{-1} is suggested to help decrease local mean age of air,[11] while 4 h^{-1} is recommended for energy savings.[12,13] However, as new information becomes available, current air change requirements for hospital wards should be re-evaluated and updated.

This study investigates the effectiveness of ventilation design strategies for general hospital wards in terms of virus removal capacity. A computational fluid dynamics (CFD)

simulation of a typical general ward of Hong Kong hospitals was conducted and three respiratory viruses, namely MERS-*CoV,* SARS-*CoV* and H1N1 influenza virus, were chosen. The findings can be used by the hospital management to minimize cross-infection risk while reducing energy consumption of ventilation.

Bioaerosol drag force

The motions of spherical and non-spherical bioaerosols in a ventilated space can be calculated using Equation (1) by integrating the force balance on the bioaerosols in terms of the drag force per unit particle mass per relative velocity F_D (N s kg^{-1} m^{-1}), where g is gravitational acceleration (m s^{-2}), F_x is the additional acceleration force per unit particle mass (N kg^{-1}) if any, v_b and v_a are the velocities of the virus and air (m s^{-1}) respectively, μ_a is the molecular viscosity of air (kg m^{-1} s^{-1}), d_b is the equivalent bioaerosol diameter (μm) and Re_b is the Reynolds number for bioaerosols in an airflow field, ρ_a is the air density (kg m^{-3}), ρ_b is the virus density (=1,100 kg m^{-3}).[14]

$$\frac{dv_b}{d\tau} = F_D(v_a - v_b) + \frac{g(\rho_b - \rho_a)}{\rho_b} + F_x;$$
$$F_D = \frac{18\mu_a}{d_b^2 \rho_b} \times \frac{C_D Re_b}{24}; Re_b = \frac{\rho_a d_b |v_b - v_a|}{\mu_a} \quad (1)$$

The drag coefficient C_D for the bioaerosols is defined by Equation (2),

$$C_D = \frac{K_D}{Re_b}; Re_b < 1 \quad (2)$$

Equations (1) and (2) are used in the CFD simulation to determine the motions of droplet nuclei under a Lagrangian scheme.

The drag constant for the bioaerosols K_D in Equation (2) is given by Equation (3),

$$K_D = \frac{d_b^2}{2} \tag{3}$$

Validity of Equation (3) was established for equivalent bioaerosol diameters d_b = 0.69 – 6.9 μm over a bioaerosol size range and further examined for *Bacteriophage Phi X174* (ATCC 13706-B1) with d_b = 0.054 μm. A combined experimental and simulation method described by Wong *et al.*[14] was performed. Before the experiment, agar plates were prepared in such a way that the entire preparation was covered by the host bacterium *Escherichia coli* (ATCC 13706). Plaque assays for bacteriophages were then conducted. The number of bacteriophages collected was measured by plate counting.[15] Figure 1 shows that Equation (3) can be extrapolated for predicting the dispersion of smaller sized bioaerosols (down to d_b = 0.054 μm). When using K_D = 0.001458 (Figure 2), the deviation between simulated and measured fractional counts inside a ventilated chamber was insignificant ($p>0.99$, *paired t-test*). The corresponding fractional bias (FB) ε_1 and normalized mean square error (NMSE) ε_2 for K_D = 0.001458 were 9.2% and –3% respectively, as illustrated in Figure 2.[16]

Using Equation (4), where the projected image area, length and width are A_b, l_1 and l_2 respectively, the equivalent bioaerosol diameter d_b can be determined from electron micrographs.[17,18] Electron micrographs of samples of MERS-*CoV*, SARS-*CoV*, H1N1 influenza virus and *Bacteriophage Phi X174* are shown in Figure 3.[19-22]

$$d_b = 2\sqrt{\frac{A_b}{\pi}}\,;\, r_{aspect} = \frac{max(l_1, l_2)}{min(l_1, l_2)} \tag{4}$$

Computational fluid dynamics (CFD) simulation

A typical semi-enclosed six-bed general ward cubicle (7.5 m (*L*) × 6 m (*W*) × 2.7 m (*H*)) employed for the CFD simulation is shown in Figure 4. As illustrated in Figure 5, there were four ceiling air inlets with mounted diffusers in the cubicle and the corridor was the air outlet. Four different air change rates (i.e. *ach* = 3, 6, 9 and 13 h^{-1}) were used, while the temperature and relative humidity of the supply air were 285 K and 80-95% respectively.[23] Six probable viral emission points, representing the positions of six patients lying on their respective beds (i.e. Man 1, Man 2, ... , Man 6), were chosen as the source locations. The viruses investigated were MERS-*CoV*, SARS-*CoV* and H1N1 influenza virus. The metabolic rate of a reclining patient was assumed to be 0.8 MET (i.e. 46.6 W m^{-2}).[24] Half of the heat (23.3 W m^{-2}) transferred from patient skin surface by convection was assumed.[25] Patient beds were set as rectangular box for the worst scenarios that airflow under the beds was fully blocked by medical equipment and relation installations. This arrangement may cause higher numbers of bioaerosol particle deposited on other patients.

The simulation of airflow field and virus dispersion inside the ward cubicle was done through the use of CFD software FLUENT 14. An Eulerian-Lagrangian framework was used to solve the gas-solid two-phase flow problem, i.e. an Eulerian scheme for the prediction of a steady-state airflow field of the ward cubicle, followed by a Lagrangian approach for the determination of virus particle movements.

In the Eulerian framework, a continuous phase of the induced airflow field was obtained with a second-order solution scheme. The renormalization group (RNG) *k*-ε turbulence model was tested to be an appropriate choice among the RANS turbulence models and therefore it was adopted to determine the air turbulence in the field. The model offered better accuracy and stability in cases of low Reynolds number and near-wall flows; and this model was found

suitable for indoor airflow simulations also.[26,27] To couple with the pressure and velocity fields, the pressure implicit with splitting of operator (PISO) algorithm was employed and the convection term was discretized using a second-order upwind scheme.

To optimize the simulation quality and speed, three reference grid sizes namely fine (i.e. 2,618 k), moderate (i.e. 1,143 k) and coarse (i.e. 1,071 k) were constructed for determining a suitable mesh size. The fine gird size was with a mesh skewness less than 0.25 to ensure an excellent cell quality. The moderate and coarse grid sizes were doubled and quadrupled by a second-order method for increasing the simulation speed. To assess the mesh quality using linear grid stretching, the asymptotic range of convergence c_{asymp} applied was based on the grid convergence indexes (GCIs) with a relative error of computed average mass flow rate ε_{rms}.[28]

If the value of c_{asymp} in Equation (5), where f_s is the safety factor, r_r is the refinement ratio and c_o is the theoretical order of convergence, is approximately 1, then the grid quality is fine.

$$GCI_{fine} = \frac{f_s|\varepsilon_{rms}|}{(r_r^{c_o} - 1)}; GCI_{coarse} = \frac{f_s|\varepsilon_{rms}|r_r^{c_o}}{(r_r^{c_o} - 1)}; c_{asymp} = \frac{GCI_{coarse}}{(GCI_{fine})r_r^{c_o}} \quad (5)$$

The GCI analysis results from the simulation were 51%, 37% and 1.187 for GCI_{coarse}, GCI_{fine} and c_{asymp} respectively.

In the CFD simulation, the transmission pathway of virus-laden respiratory droplets expelled by sneezing was predicted (i.e. d_i, initial virus-laden respiratory droplets diameter = 8.3 μm, v_b = 50 m s^{-1}, ρ_b = 1,100 kg m^{-3} and n_s, amount of virus particles expelled by sneezing = 10,000 virus particles).[14,29,30,31] General patient wards are recommended at an relative humidity 30-60%.[6] Within a short period of time (<0.1 s) after emission, the droplets would evaporate to droplet nuclei.[32] These nuclei were the dried-out residual of droplets possibly

containing infectious pathogens.[33] Only a small proportion (<10%) of the virus-laden droplets of a total number of 73,000 - 1,000,000 droplets expelled by a vigorous sneeze was assumed; neither aggregation with other particles nor cluster of virus would be formed from the dried-out nuclei at such low concentration.[34,35] Lipid-enveloped human coronavirus 299E would remain alive by a half-life of 67 hours at an RH of 50% and an air temperature of 20°C.[36] The survival rate of the three virus was expected to be 100% in simulation times less than 100s. Other details for simulations and boundary conditions are summarized in Table 1.

Table 2 exhibits the equivalent bioaerosol diameters d_b for the virus droplet nuclei examined in this study. A one-way coupling was applied in the prediction to prevent the effect of particles on the continuum airflow. Each virus particle was tracked separately for its position and velocity by a discrete phase model (DPM). A previous study confirmed that the isotropic discrete random walk (DRW) model was effective and accurate in modelling bioaerosol dispersion and distribution due to turbulent fluctuations in the flow.[37] For a coagulation effect of bioaerosol particles in this study, a very low volume fraction (<3,000 cm^{-3}) was kept in the ward cubicle to reduce collisions of the virus particles in a turbulent flow.[38,39] Both the amounts of virus particles exhausted to the corridor n_e and deposited on the ward room surfaces n_d were counted.[40] A perfect sink boundary condition was applied to the ward room surfaces in order that the virus particles impinged onto the solid surfaces would be perfectly trapped with no reflection and desorption.[41]

Deposited ratio

Direct contact with the viruses deposited on ward room surfaces and inhalation of the viruses suspended in the air are two potential transmission routes of virus-laden airborne particles expelled by sneezing.[42] The deposited ratio r_d is selected as a measure for evaluating the contact transmission; the movement of sneeze particles can be represented by Equation (6),

where r_e is the exhausted ratio for particles exhausted to the corridor and r_a is the elapsed ratio for particles suspended in the air,

$$r_e + r_a + r_d = 1 \tag{6}$$

Ratios r_e, r_a and r_d are given by Equation (7), where n_s, n_e, n_a and n_d are the amounts of virus particles expelled by sneezing, exhausted to the corridor, elapsed (with respect to air suspension) and deposited on ward room surfaces respectively,

$$r_e = \frac{n_e}{n_s};\ r_a = \frac{n_a}{n_s};\ r_d = \frac{n_d}{n_s} \tag{7}$$

Figure 6 graphs the ratios r_e, r_a and r_d against time. The elapsed time τ_a (s) required for all virus particles to be exhausted or to deposit on ward room surfaces can be determined from the figure as represented by Equation (8).

$$r_a(\tau_a) = 0 \tag{8}$$

Results and discussion

Airflow field

Figure 7 shows the simulated temperature, air velocity distribution and the flow pathlines inside the ward with *ach* = 6 h^{-1}. An increasing temperature gradient was found from the ceiling to the floor inside the ward. As shown in Figure 7(a), warmer air observed near the patients (due to body temperature) was diffusing out to the corridor. The average room temperature was about 296 K. Stagnant air, with velocity less than 0.05 m s^{-1}, was found near the window side (i.e. Wall 2) and the lower floor level (i.e. below 0.3 m) as presented in Figure 7(b). The flow pathlines in Figure 7(c) confirm that there was insufficient ventilation to remove

any virus particles in those areas. Generally, the airflow direction was from the inner part of the ward to the corridor.

Cross-infection from surface deposition

Figure 8 indicates the potential risks of cross-infection with MERS-*CoV* through air pathways from different infected patients inside the ward. Patients staying on the same side of an infected patient, especially the one located next to the corridor (i.e. Man 1 or Man 2), would have a higher chance of cross-infection. Two different virus pathway flows in the simulation due to the asymmetric diffuser locations are highlighted. Cases exhibited in Figures 8(c) and (e) show the virus moved along floor surface of the ward but in cases shown in Figures 8(d) and (f), virus would pass over nearby patients' heads, then flew to the corridor.

Figure 9 exhibits the values of deposited ratio r_d and exhausted ratio r_e of MERS-*CoV* on all surfaces inside the ward with *ach* = 3, 6, 9 and 13 h^{-1}. The results suggested that the deposition of virus particles was dependent on the location of an infected patient and the virus particles would deposit mainly back on the source patient. If the infected patient was located near the corridor (i.e. Man 1 or Man 2), the virus particles would likely be exhausted to the corridor. On the contrary, if the infected patient was located in the inner part of the ward (i.e. Man 5 or Man 6), the possibility that the virus particles would deposit on the wall surfaces or other patients was higher.

Moreover, more virus particles would remain on the sneezing patient at lower air change rates, especially for *ach* = 6 h^{-1} (r_d > 0.7 for all source locations), whereas more virus particles would deposit on the wall surfaces or be exhausted to the corridor at higher air change rates (i.e. *ach* = 9 h^{-1} or 13 h^{-1}). Although the possibility of cross-infection among patients and healthcare workers (HCWs) was lower at a higher *ach* (up to 9 h^{-1}), HCWs should clean all patients and ward surfaces regularly, regardless of *ach*.

Cross-infection from inhalation

Figure 10 graphs the MERS-*CoV* removal processes in the ward for the six source locations (i.e. Man 1, Man 2, ... , Man 6) and *ach* = 3, 6, 9 and 13 h^{-1}. The results showed that virus particles from an infected patient located near the corridor were likely to be exhausted to the corridor. Meanwhile, lower r_e (i.e. higher r_d) and longer elapsed time τ_a were associated with the MERS-*CoV* particles from the sneezing of an infected patient located in the inner part of the ward, and that indicated a higher risk of MERS-*CoV* infection through direct inhalation of particles or indirect inhalation of resuspended particles. Besides, higher r_e and shorter τ_a were found with increasing air change rates. If *ach* was increased from 3 h^{-1} to 13 h^{-1}, τ_a could be shortened by more than 30 s and the risk of cross-infection from inhalation could be effectively lowered. Similar dispersion and deposition results were observed for SARS-*CoV* and H1N1 influenza virus particles and pictorialized in Figures 11 and 12. The simulation performances demonstrated that virus particles with a relatively small equivalent bioaerosol diameter ($d_b \sim 0.1$ μm) had similar particle dispersion and deposition characteristics in a general hospital ward.

Figure 13 plots the average elapsed time τ_a against *ach* for MERS-*CoV*, SARS-*CoV* and H1N1 influenza virus. The results showed that τ_a could be significantly shortened by increasing the air change rate in the ward ($R^2 > 0.9$). However, the threshold or optimal τ_a in the ward could not be determined as the infectious doses (ID50) of the three viruses varied in wide ranges (i.e. 180, 1,800 and 180 virus particles for MERS-*CoV*, SARS-*CoV* and H1N1 influenza virus respectively to cause a 50% infection).[43-45] Using the ASHRAE standard for an AIIR (i.e. *ach* = 12 h^{-1}) as a safety measure,[6] the corresponding elapsed time was about 34 s. When applying the ASHRAE (*ach* = 4 h^{-1}) and CIBSE (*ach* = 6 h^{-1}) standards for a general patient room, the average values of τ_a were found to be 70 s and 61 s respectively.[6,8] The elapsed time doubled when *ach* dropped from 12 h^{-1} to 4 h^{-1}, and thus doubling the potential inhalation risk. Based

on the median value in accordance with both ASHRAE and CIBSE standards, the maximum *ach* in a general hospital ward should be 9 h^{-1} (τ_a = 48 s) for the needs of maximizing energy efficiency and minimizing infection risk. Furthermore, it should be noted for other means to minimize the possibility of cross-contamination in hospital wards, such as the installation of Ultraviolet Germicidal Irradiation (UVGI) lamps for the destruction of viral nucleic acids.[46]

Conclusion

This study should provide a useful source of reference for the hospital management to mitigate the risk of infection with MERS or other airborne transmitted viruses through better ventilation design strategies. The results of this study demonstrated that the location of an infected patient would affect the infection risks to other occupants and HCWs inside the same hospital ward, and an increased air change rate in the ward could reduce the risk of infection from direct contact and inhalation. For a typical semi-enclosed six-bed general ward of Hong Kong hospitals, an air change rate of 9 h^{-1} could effectively minimize the deposition and floating time of respiratory virus particles while maximizing energy efficiency. In order to minimize the possibility of cross-contamination in hospital wards, installation of UVGI lamps is also recommended.

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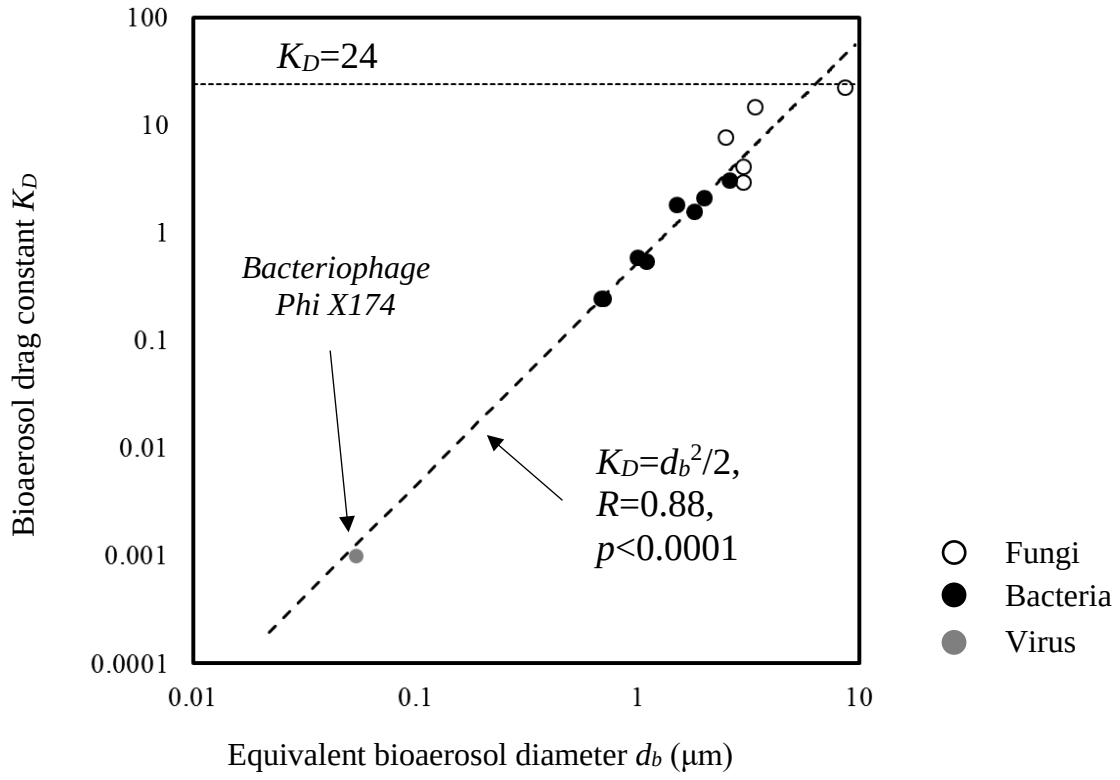


Figure 1. Bioaerosol drag constant K_D against equivalent bioaerosol diameter d_b

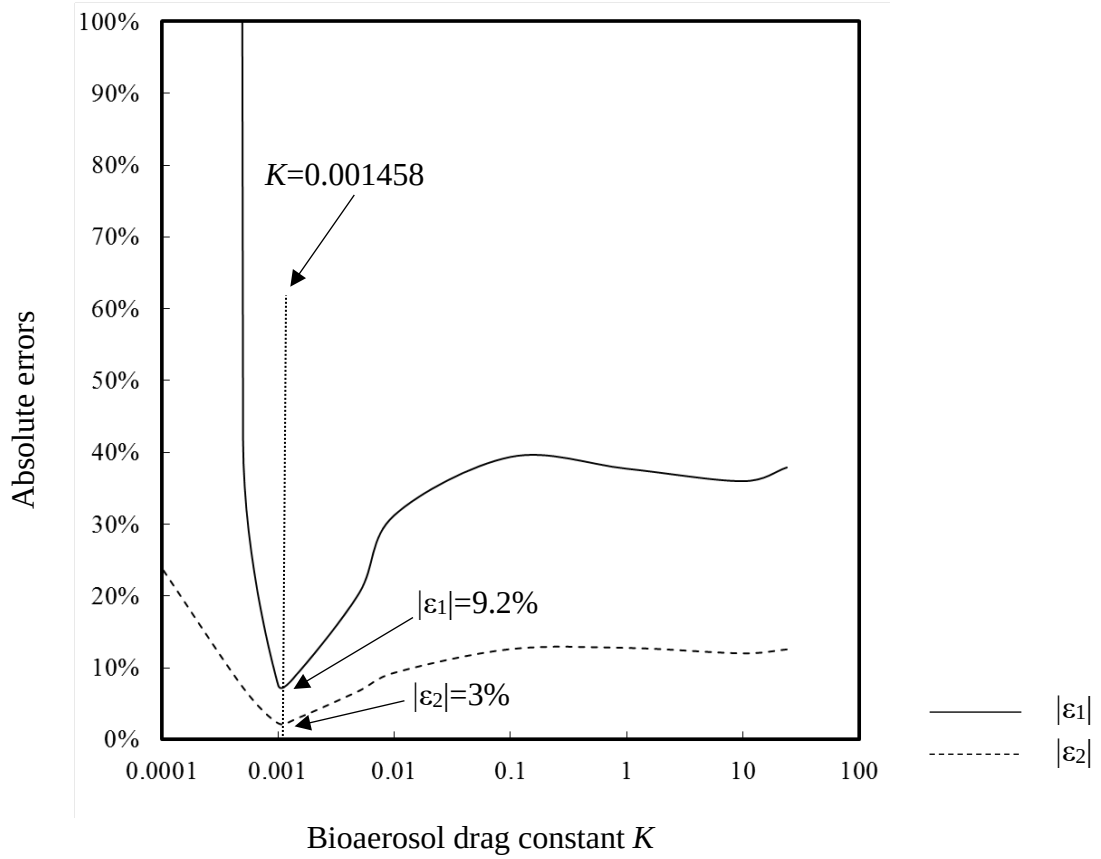


Figure 2. Bioaerosol drag constants and absolute errors for *Bacteriophage Phi X174*

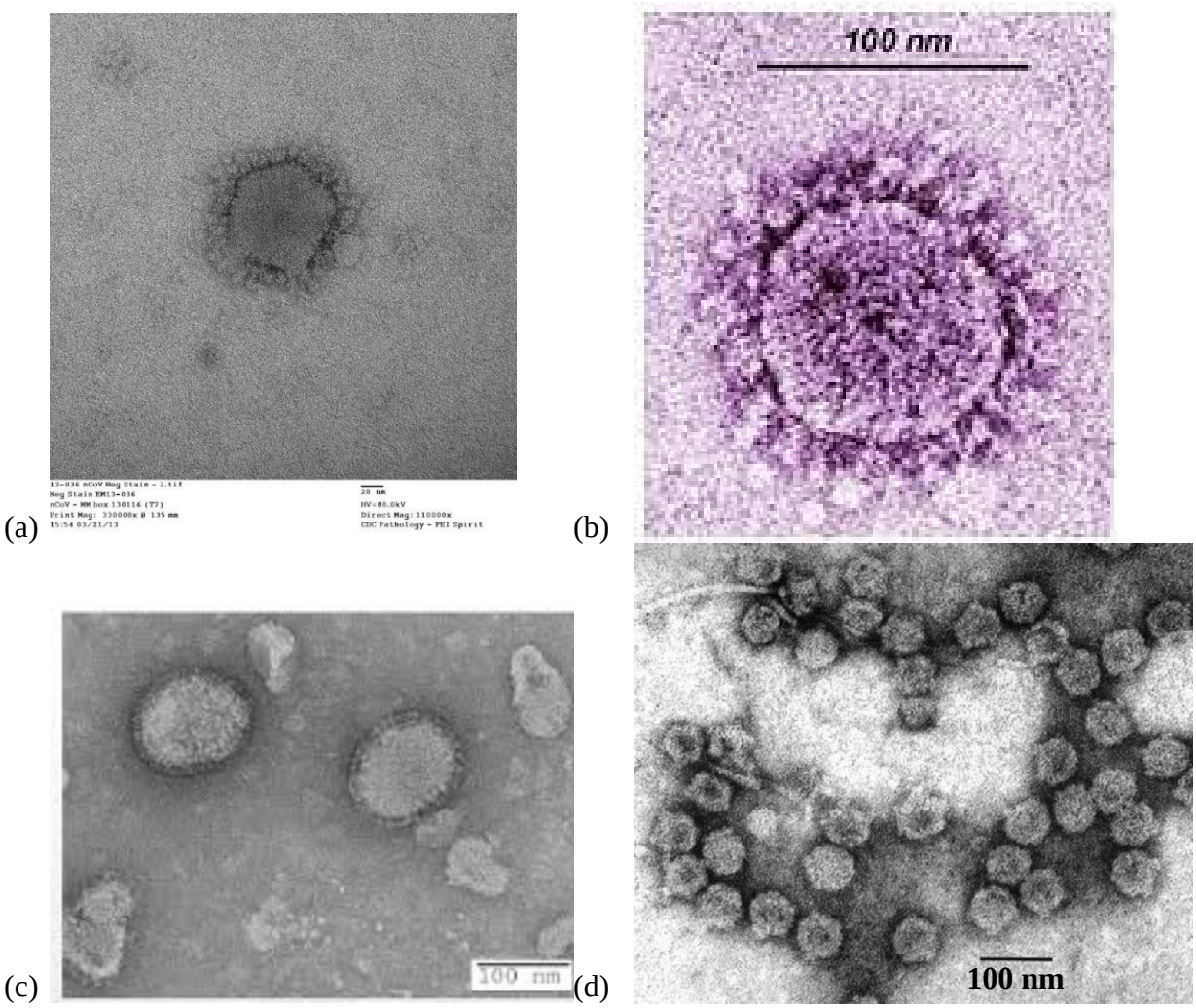


Figure 3. Electron micrograph of reference viruses. (a) MERS-*CoV*[19] (b) SARS-*CoV*[20] (c) H1N1 influenza virus[21] and (d) *Bacteriophage Phi X174*[22]

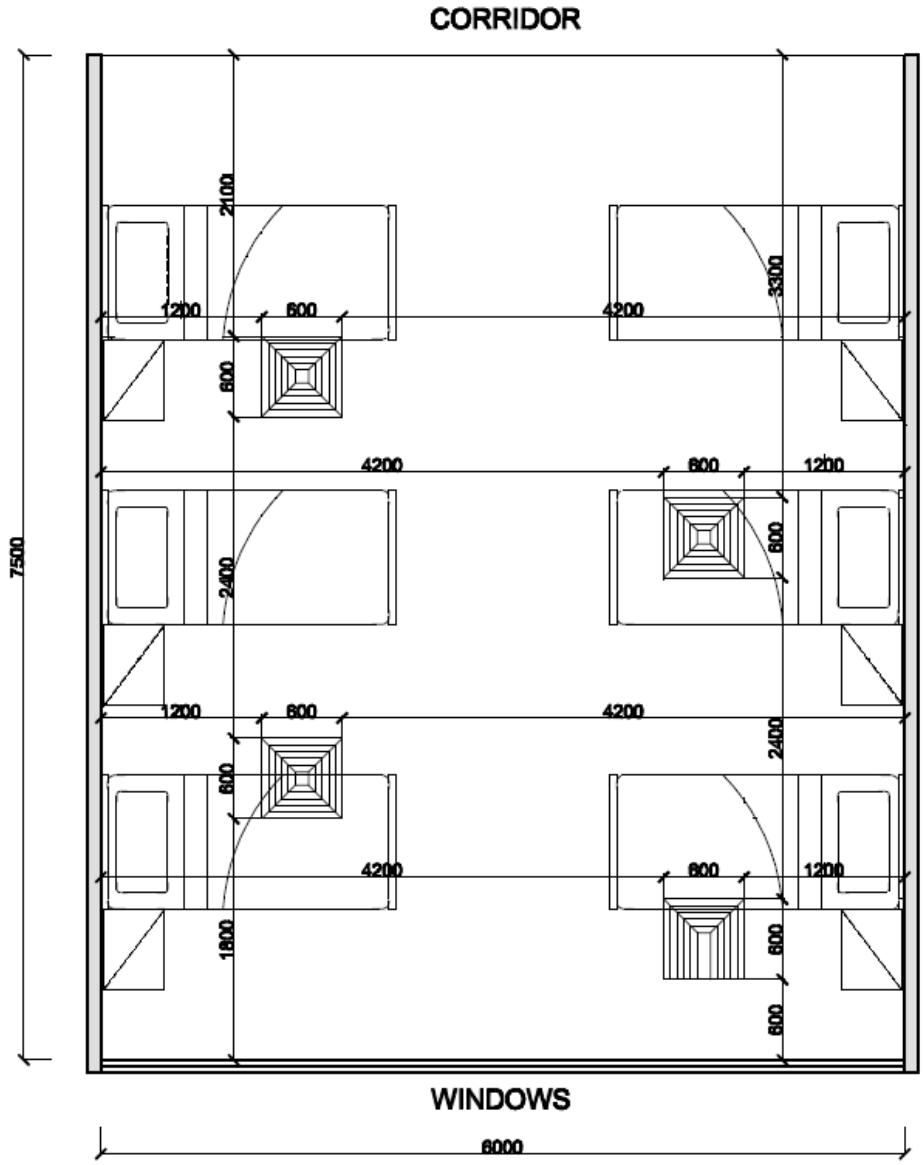


Figure 4. A six-bed general ward cubicle (dimensions in mm)

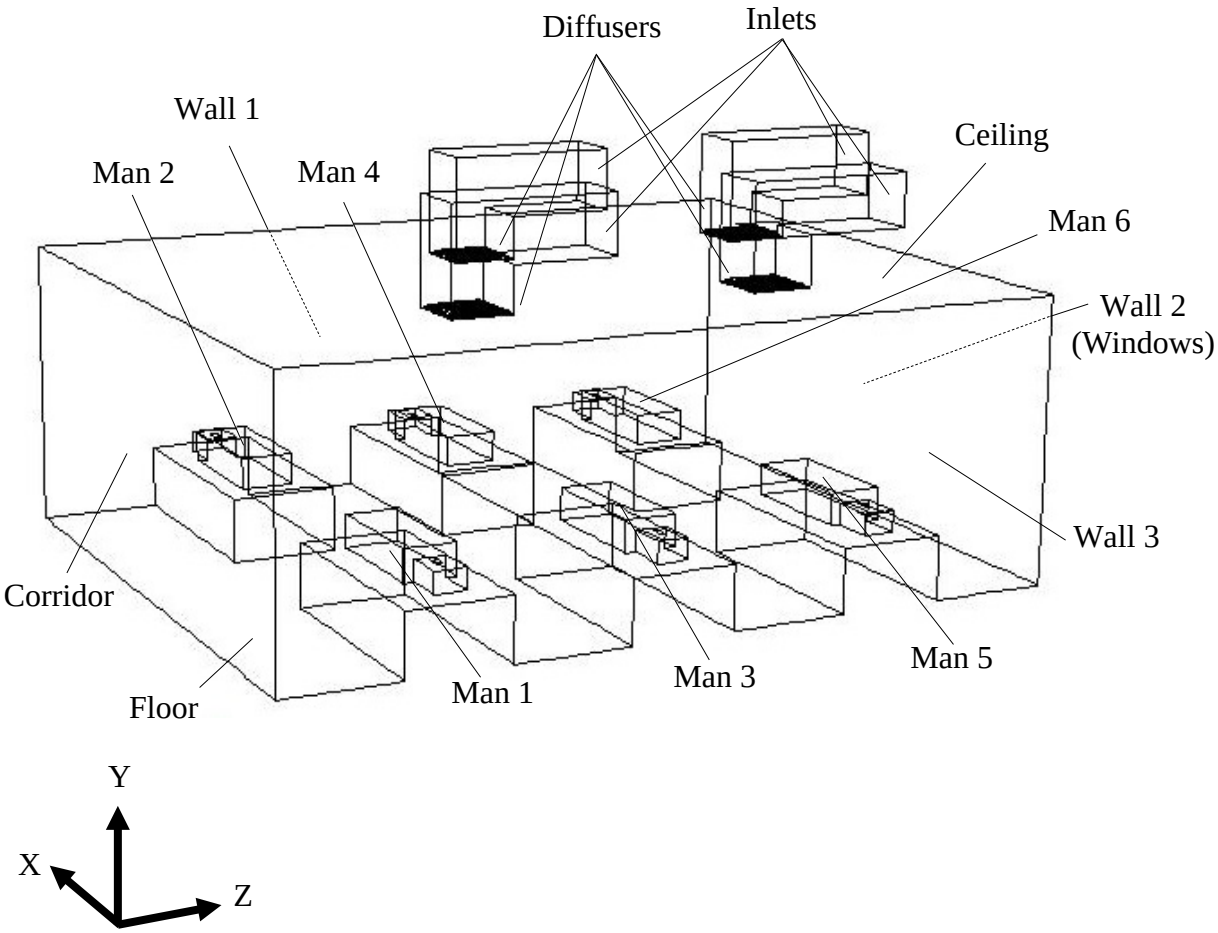


Figure 5. CFD configurations of a six-bed general ward cubicle

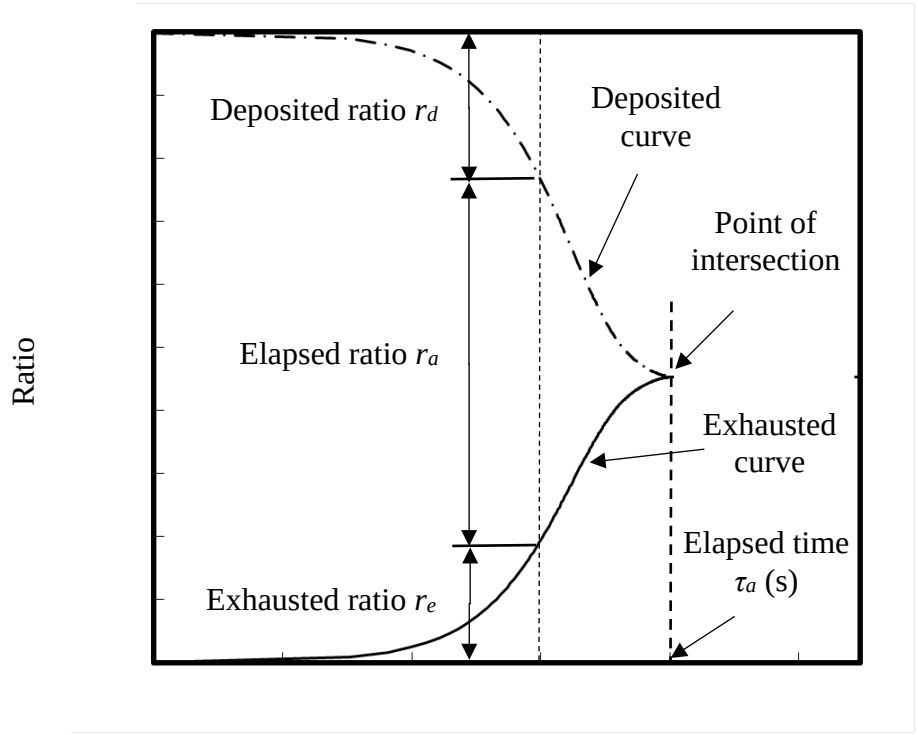
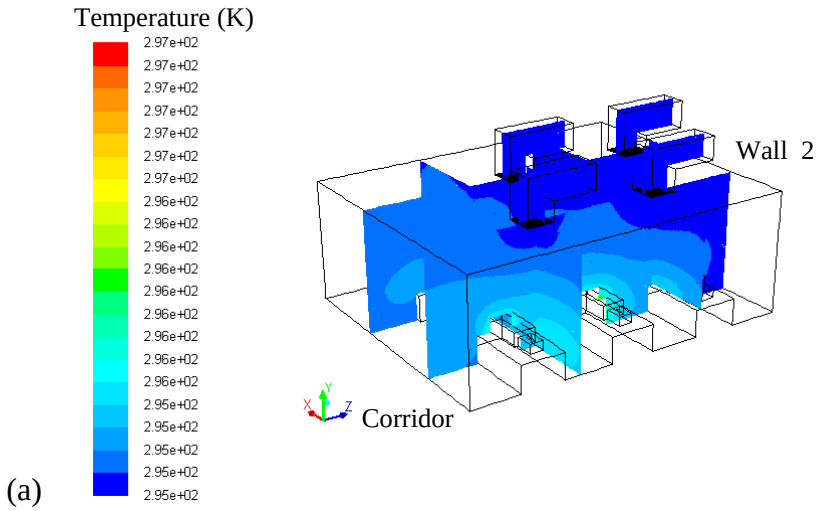
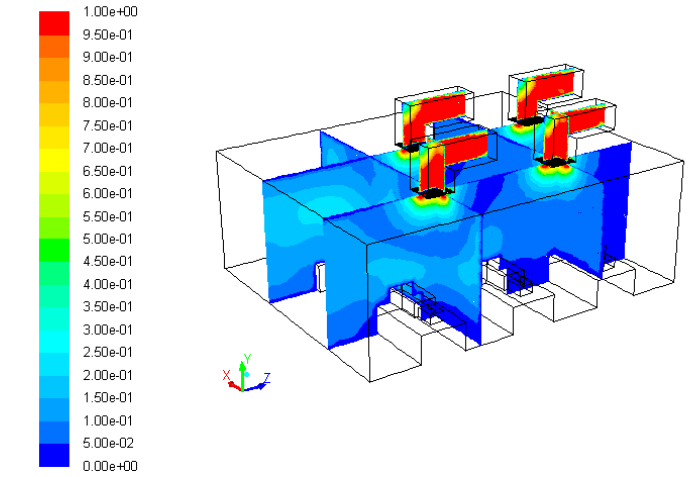


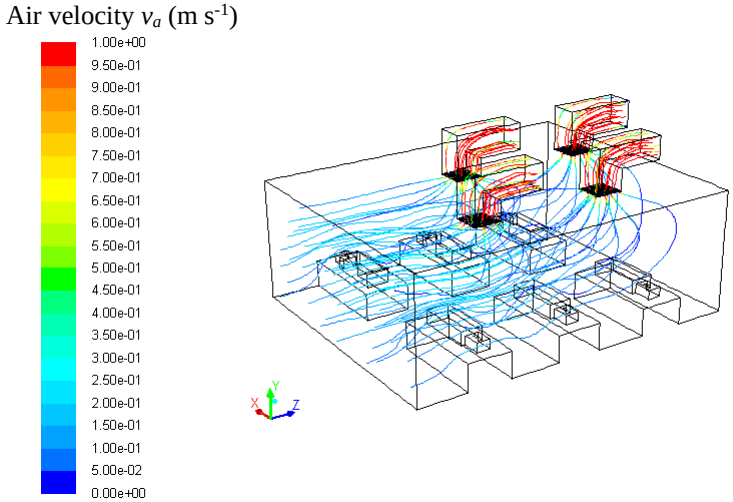
Figure 6. Description of the bioaerosol removal process in a general ward



(a)



(b)



(c)

Figure 7. Simulation results of the ward with *ach* = 6 h^{-1}. (a) Temperature distribution, (b) Air velocity distribution, (c) Flow pathlines from air supply inlets

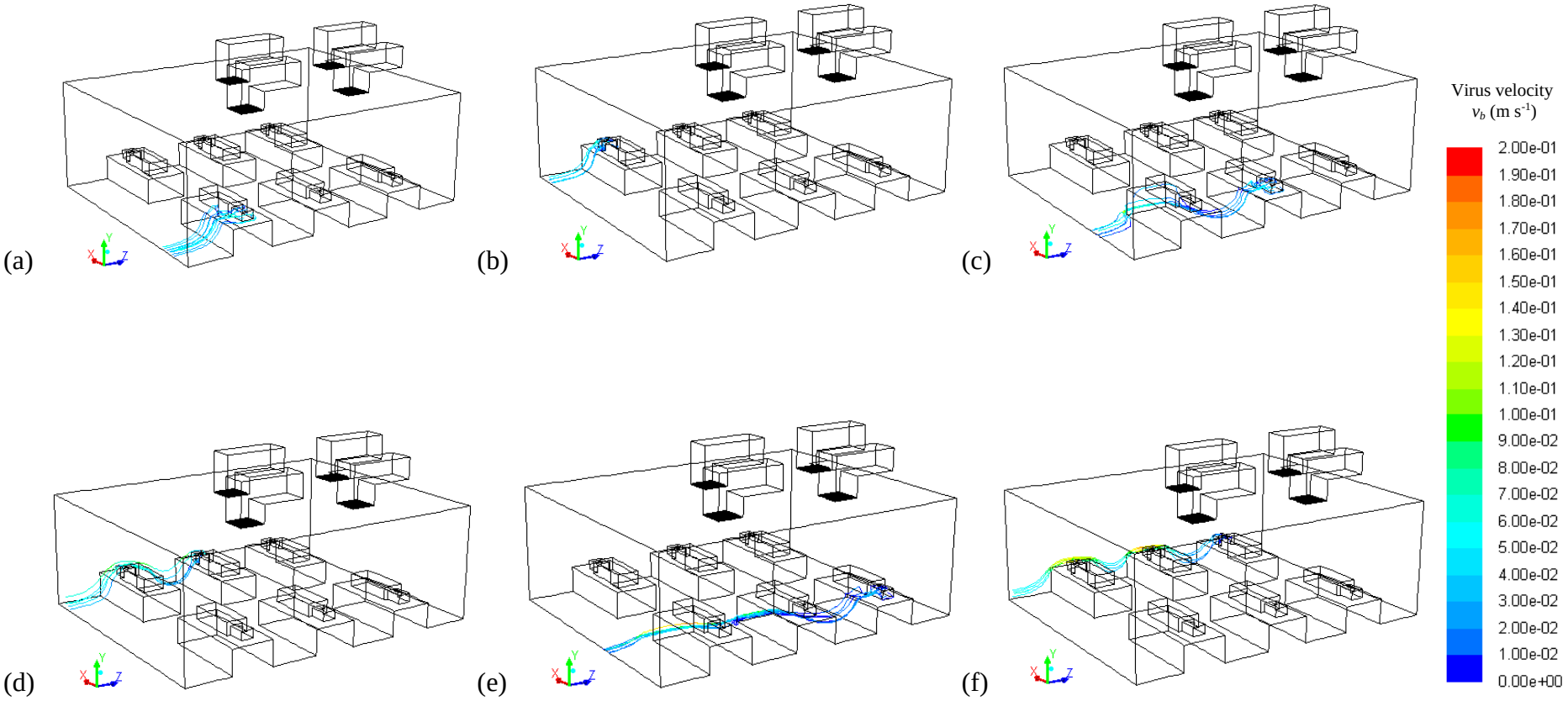
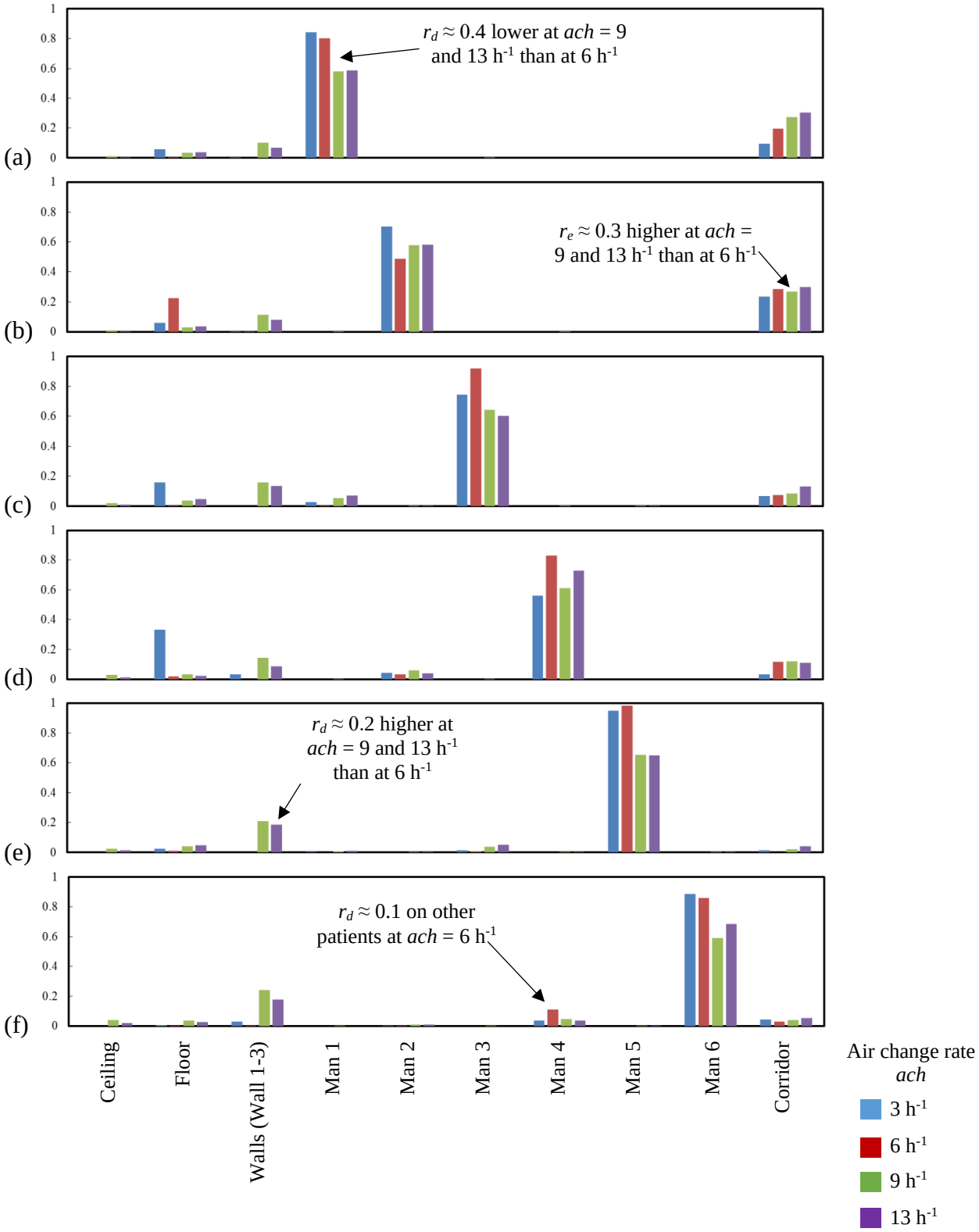


Figure 8. MERS-*CoV* pathways for 6 source locations with *ach* = 6 h^{-1}. (a) Man 1, (b) Man 2, (c) Man 3, (d) Man 4, (e) Man 5 and (f) Man 6



x-axis: Deposition surfaces

y-axis: Deposited ratio r_d (on surfaces) or exhausted ratio r_e (to corridor)

Figure 9. Deposited and exhausted ratios of MERS-*CoV* for 6 source locations. (a) Man 1, (b) Man 2, (c) Man 3, (d) Man 4, (e) Man 5 and (f) Man 6

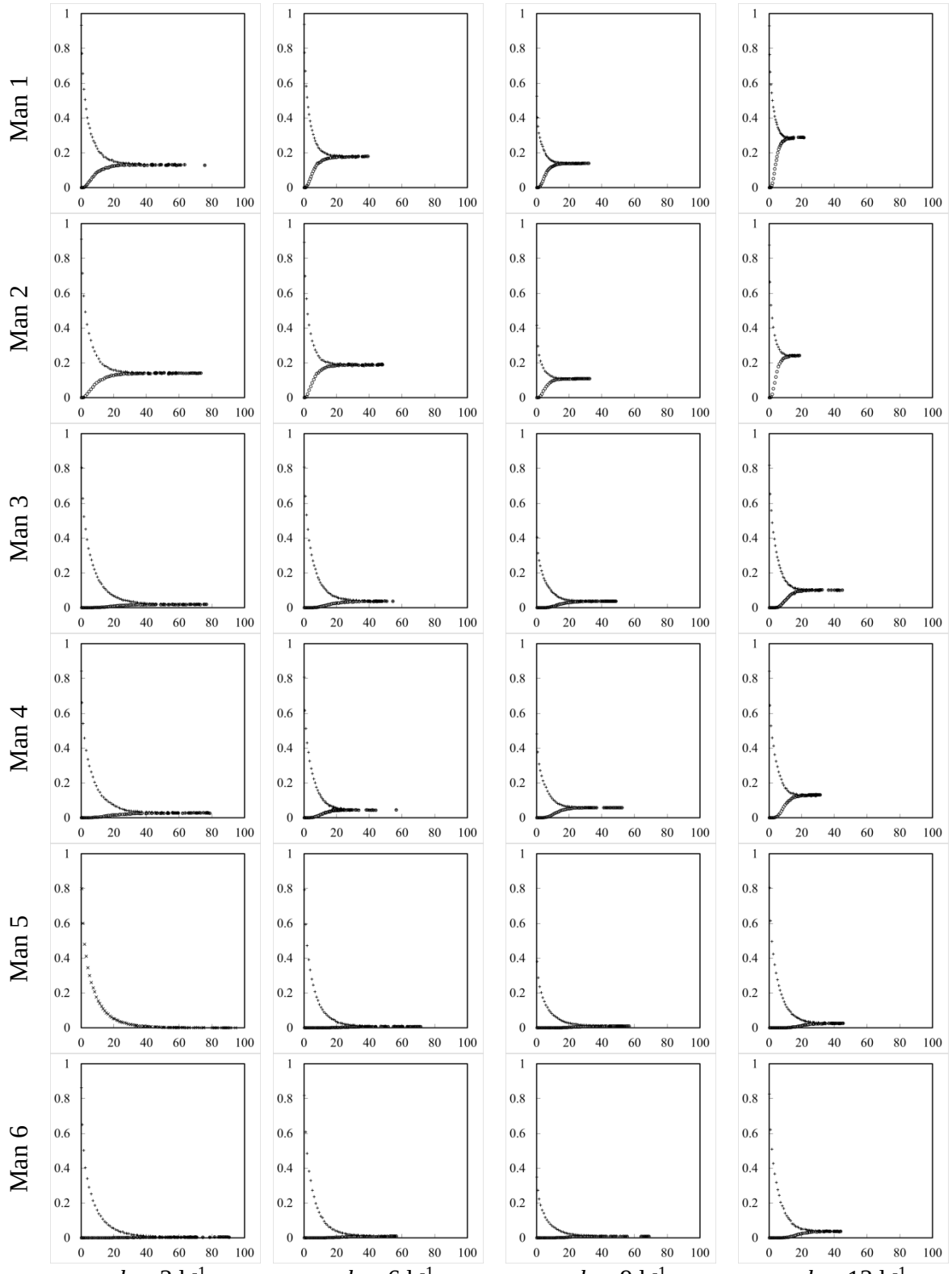


Figure 10. MERS-*CoV* removal processes for different source locations and air change rates

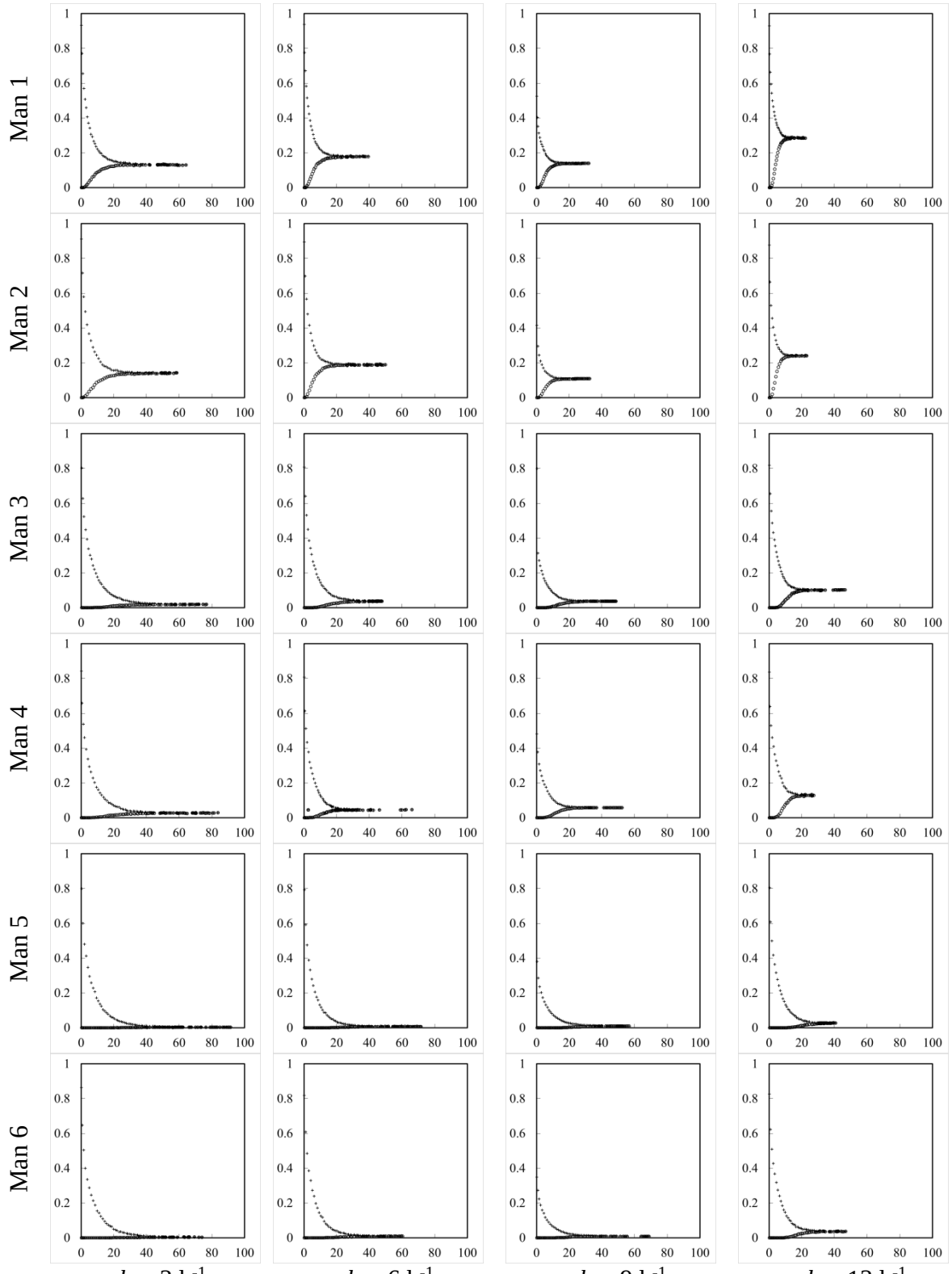


Figure 11. SARS-*CoV* removal processes for different source locations and air change rates

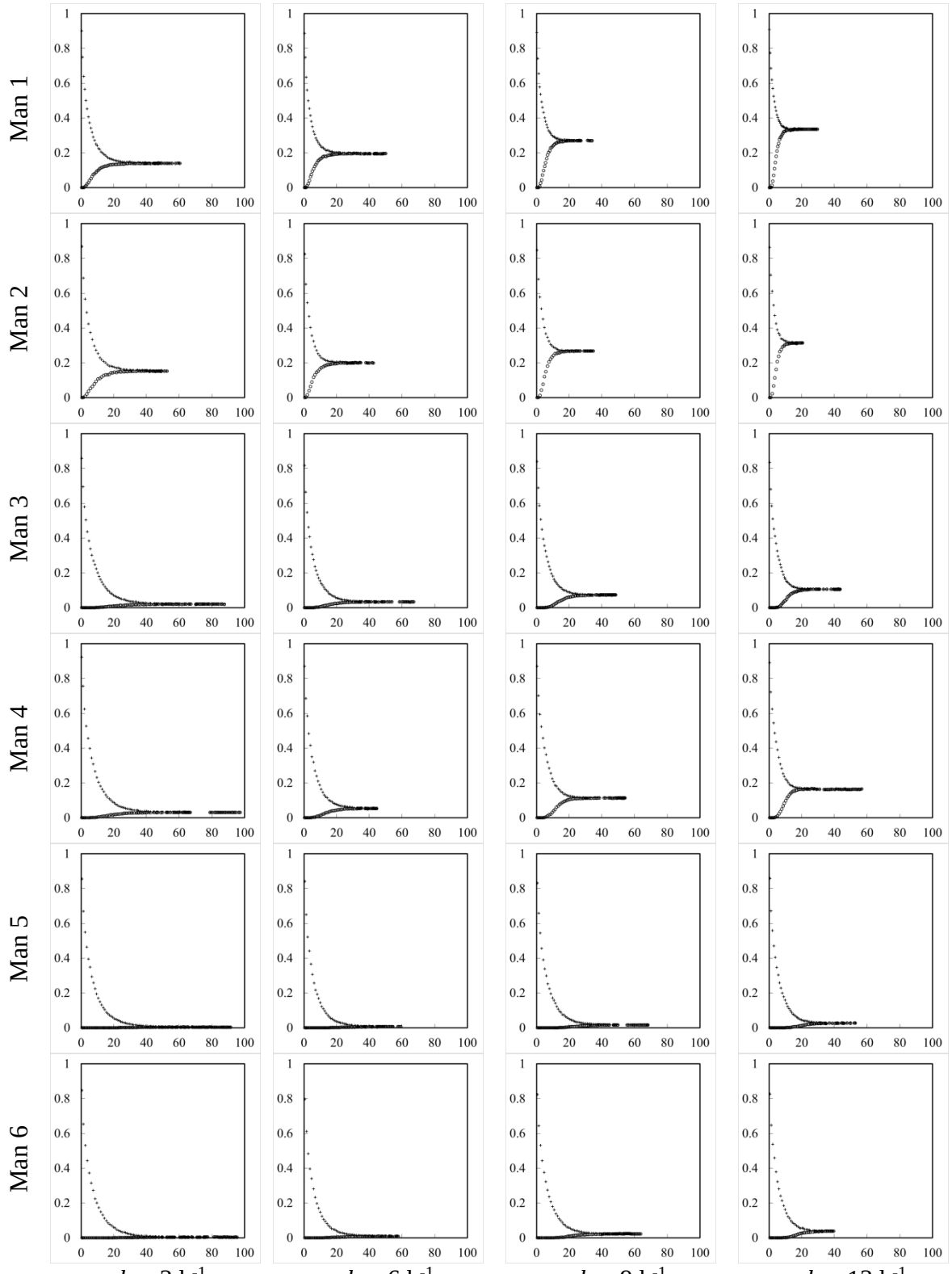


Figure 12. H1N1 influenza virus removal processes for different source locations and air change rates

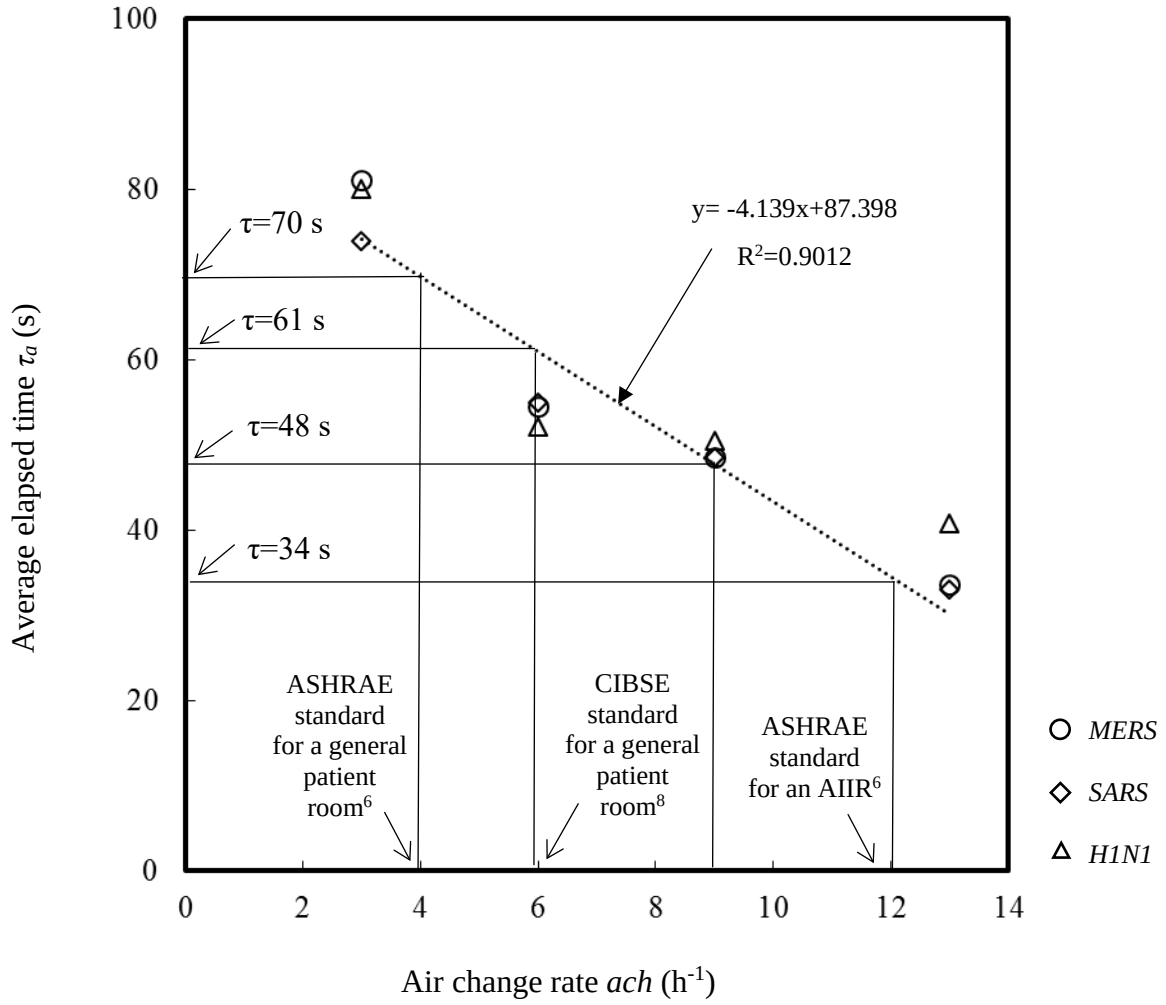


Figure 13. Average elapsed time τ_a with design standards

Table 1. CFD simulations and boundary conditions.

Computational domain	7.5 m (L) × 6 m (W) × 2.7 m (H), RNG k-ε turbulence model, standard wall function.
Mesh configuration	1,143,766 cells
Total supply airflow rate	1.24 kg s^{-1} (for ach=3), 2.48 kg s^{-1} (for ach=6), 3.72 kg s^{-1} (for ach=9) and 5.37 kg s^{-1} (for ach=13), 285K (air temperature)
Each inlet airflow rate (0.6 m × 0.6 m)	0.31 kg s^{-1} (for ach=3), 0.62 kg s^{-1} (for ach=6), 0.93 kg s^{-1} (for ach=9) and 1.34 kg s^{-1} (for ach=13), 285K (air temperature)
Four diffuser (0.6 m × 0.6 m)	Four-way spread-type, supply jets had an angle of 15° from ceiling, adiabatic and reflect boundary type.
Corridor (6 m × 2.7 m)	Pressure-outlet, 295 K (backflow temperature), adiabatic, escape boundary type.
Walls, ceiling, floor and beds	No slip wall boundary, 295 K (surface temperature), adiabatic, trap boundary type.
Patients	No slip wall boundary, 23.3 W m^{-2}, trap boundary.
Patient mouths (0.05 m × 0.05 m)	Single-shot release with an upward exhalation velocity of v_b=50 m s^{-1}, initial virus-laden respiratory droplet diameter d_i=8.3 μm, n_s=10,000 virus particles, density of bioaerosol particle ρ_b=1,100 kg m^{-3}

Table 2. Virus information

Species	ATCC	Equivalent bioaerosol diameter d_b (µm)*	Aspect ratio r_{aspect}	Drag constant K_D	Evaporation time at 0% RH (s)	Evaporation time at 50% RH (s)	Evaporation time at 90% RH (s)
MERS-*CoV*	-	0.167±0.012	1.27	0.013945	3.48×10^{-2}	9.55×10^{-2}	1.82×10^{-1}
SARS-*CoV*	-	0.1375±0.009	1.052	0.009453	3.48×10^{-2}	9.55×10^{-2}	1.82×10^{-1}
H1N1 influenza virus	-	0.124±0.0001	1.203	0.0199	3.48×10^{-2}	9.55×10^{-2}	1.82×10^{-1}
Bacteriophage Phi X174	13706-B1	0.054±0.014	1.012	0.001458	-	-	-

*Standard errors shown

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