Ventilation of general hospital wards for mitigating infection risks of three kinds of viruses including Middle East respiratory syndrome coronavirus

H.C. Yu, K.W. Mui, L.T. Wong and H.S. Chu

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)

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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 2012/2013 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 Centers 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

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quality and energy efficiency, general hospital wards have been designed to meet certain air change requirements.\(^2\)\(^6\)\(^7\)\(^8\)\(^9\)\(^10\) 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, \(\mu_a\) is the molecular viscosity of air (kg m\(^{-1}\) s\(^{-1}\)), \(d_b\) is the equivalent bioaerosol diameter (\(\mu\)m), \(Re_b\) is the Reynolds number for bioaerosols in an airflow field, \(\rho_a\) is the air density (kg m\(^{-3}\)) and \(\rho_b\) is the virus density (=1100 kg m\(^{-3}\)).\(^14\)

\[
\frac{dv_b}{dt} = F_D(v_a - v_b) + g(\rho_b - \rho_a) + F_x; \\
F_D = \frac{18\mu_a}{d_b\rho_b} \times \frac{C_D Re_b}{24}; \\
Re_b = \frac{\rho_a d_b |v_b - v_a|}{\mu_a}
\]

(1)

The drag coefficient \(C_D\) for the bioaerosols is defined by equation (2)

\[
C_D = \frac{K_D}{Re_b}; \quad Re_b < 1
\]

(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)

\[
K_D = \frac{d_b^2}{2}
\]

(3)

Validity of equation (3) was established for equivalent bioaerosol diameters \(d_b = 0.69 - 6.9\ \mu\)m over a bioaerosol size range and further examined for Bacteriophage Phi X174 (ATCC 13706-B1) with \(d_b = 0.054\ \mu\)m. 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 \( d_b = 0.054 \mu m \)). When using \( K_D = 0.001458 \) (Figure 2), the deviation between simulated and measured fractional counts inside a ventilated chamber was insignificant \((p > 0.99, \text{ paired } t\text{-test})\). The corresponding fractional bias (FB) \( \varepsilon_1 \) and normalized mean square error (NMSE) \( \varepsilon_2 \) 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.\(^{19-22} \)

\[
d_b = 2 \sqrt{\frac{A_b}{\pi}}, \quad \text{aspect} = \frac{\max(l_1, l_2)}{\min(l_1, l_2)}
\]

**CFD simulation**

A typical semi-enclosed six-bed general ward cubicle (7.5 m \((L) \times 6\ m \((W) \times 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 \((i.e. \text{ach} = 3, 6, 9 \text{ and } 13 \text{ h}^{-1})\) were used, while the temperature and relative humidity (RH) 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 \(\text{(i.e. Man 1, Man 2, ..., Man 6)}\), 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 \((i.e. 46.6 \text{ W m}^{-2})\).\(^{24} \) Half of the heat \((23.3 \text{ W m}^{-2})\) 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) $k$-$\varepsilon$ 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.\textsuperscript{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. 2618 k), moderate (i.e. 1143 k) and coarse (i.e. 1071 k) were constructed for determining a suitable mesh size. The fine grid
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 \( c_{\text{asymp}} \) applied was based on the grid convergence indexes (GCIs) with a relative error of computed average mass flow rate \( \varepsilon_{\text{rms}} \).

If the value of \( c_{\text{asymp}} \) 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.

\[
GCI_{\text{fine}} = \frac{f_s |\varepsilon_{\text{rms}}|}{(r_r^o - 1)}; \quad GCI_{\text{coarse}} = \frac{f_s |\varepsilon_{\text{rms}}|}{(r_r^o - 1)}; \quad c_{\text{asymp}} = \frac{GCI_{\text{coarse}}}{GCI_{\text{fine}}}r_r^o.
\]  

(5)

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

In the CFD simulation, the transmission pathway of virus-laden respiratory droplets expelled by sneezing was predicted (i.e. \( d_v \) initial virus-laden respiratory droplets diameter = 8.3 \( \mu \)m, \( v_b \) = 50 m s\(^{-1}\), \( \rho_b \) = 1100 kg m\(^{-3}\) and \( n_v \) amount of virus particles expelled by sneezing = 10,000 virus particles). \(^{14,29-31}\) General patient wards are recommended at an RH 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 h 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 100 s. 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 (<3000 cm\(^{-3}\)) was kept in the ward cubicle to reduce collisions of the virus particles in

\[
\begin{align*}
GCI_{\text{fine}} & = \frac{f_s |\varepsilon_{\text{rms}}|}{(r_r^o - 1)}; \\
GCI_{\text{coarse}} & = \frac{f_s |\varepsilon_{\text{rms}}|}{(r_r^o - 1)}; \\
c_{\text{asymp}} & = \frac{GCI_{\text{coarse}}}{GCI_{\text{fine}}}r_r^o.
\end{align*}
\]
a turbulent flow. Both the amounts of virus particles exhausted to the corridor \( n_e \) and deposited on the ward room surfaces \( n_d \) were counted. A perfect sink boundary condition was applied to the ward room surfaces in order that the virus particles impinging on the solid surfaces would be perfectly trapped with no reflection and desorption.

### 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. 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 \( n_s \) is the exhausted ratio for particles exhausted to the corridor and \( n_a \) is the elapsed ratio for particles suspended in the air.

\[
  r_e + r_a + r_d = 1 
\]

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

### Table 1. CFD simulations and boundary conditions.

<table>
<thead>
<tr>
<th>Computational domain</th>
<th>7.5 m (L) ( \times 6 ) m (W) ( \times 2.7 ) m (H)</th>
<th>RNG k-e turbulence model, standard wall function.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mesh configuration</td>
<td>1,143,766 cells</td>
<td></td>
</tr>
<tr>
<td>Total supply airflow rate</td>
<td>1.24 kg s(^{-1}) (for ( \text{ach} = 3 )), 2.48 kg s(^{-1}) (for ( \text{ach} = 6 )), 3.72 kg s(^{-1}) (for ( \text{ach} = 9 )) and 5.37 kg s(^{-1}) (for ( \text{ach} = 13 )), 285 K (air temperature)</td>
<td></td>
</tr>
<tr>
<td>Each inlet airflow rate (0.6 m ( \times 0.6 ) m)</td>
<td>0.31 kg s(^{-1}) (for ( \text{ach} = 3 )), 0.62 kg s(^{-1}) (for ( \text{ach} = 6 )), 0.93 kg s(^{-1}) (for ( \text{ach} = 9 )) and 1.34 kg s(^{-1}) (for ( \text{ach} = 13 )), 285 K (air temperature)</td>
<td></td>
</tr>
<tr>
<td>Four diffuser (0.6 m ( \times 0.6 ) m)</td>
<td>Four-way spread-type, supply jets had an angle of 15(^{\circ}) from ceiling, adiabatic and reflect boundary type.</td>
<td></td>
</tr>
<tr>
<td>Corridor (6 m ( \times 2.7 ) m)</td>
<td>Pressure-outlet, 295 K (backflow temperature), adiabatic, escape boundary type.</td>
<td></td>
</tr>
<tr>
<td>Walls, ceiling, floor and beds</td>
<td>No slip wall boundary, 295 K (surface temperature), adiabatic, trap boundary type.</td>
<td></td>
</tr>
<tr>
<td>Patients</td>
<td>No slip wall boundary, 23.3 W m(^{-1}), trap boundary.</td>
<td></td>
</tr>
<tr>
<td>Patient mouths (0.05 m ( \times 0.05 ) m)</td>
<td>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 \mu m ), ( n_s = 10,000 ) virus particles, density of bioaerosol particle ( \rho_b = 1100 ) kg m(^{-3}).</td>
<td></td>
</tr>
</tbody>
</table>

RNG: renormalization group.

### Table 2. Virus information.

<table>
<thead>
<tr>
<th>Species</th>
<th>ATCC</th>
<th>Equivalent bioaerosol diameter ( d_b ) (( \mu m )) ( ^{a} )</th>
<th>Aspect ratio ( t_{aspect} )</th>
<th>Drag constant ( K_D )</th>
<th>Evaporation time at 0% RH (s)</th>
<th>Evaporation time at 50% RH (s)</th>
<th>Evaporation time at 90% RH (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MERS-CoV</td>
<td>–</td>
<td>0.167 ± 0.012</td>
<td>1.27</td>
<td>0.013945</td>
<td>3.48 \times 10^{-2}</td>
<td>9.55 \times 10^{-2}</td>
<td>1.82 \times 10^{-1}</td>
</tr>
<tr>
<td>SARS-CoV</td>
<td>–</td>
<td>0.1375 ± 0.0009</td>
<td>1.052</td>
<td>0.009453</td>
<td>3.48 \times 10^{-2}</td>
<td>9.55 \times 10^{-2}</td>
<td>1.82 \times 10^{-1}</td>
</tr>
<tr>
<td>H1N1 influenza virus</td>
<td>–</td>
<td>0.124 ± 0.0001</td>
<td>1.203</td>
<td>0.0199</td>
<td>3.48 \times 10^{-2}</td>
<td>9.55 \times 10^{-2}</td>
<td>1.82 \times 10^{-1}</td>
</tr>
<tr>
<td>Bacteriophage Phi X174</td>
<td>13706-B1</td>
<td>0.054 ± 0.014</td>
<td>1.012</td>
<td>0.001458</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
</tbody>
</table>

MERS-CoV: Middle East respiratory syndrome coronavirus; SARS-CoV: severe acute respiratory syndrome coronavirus. \( ^{a} \)Standard errors shown.
Figure 7. Simulation results of the ward with $ach = 6\, \text{h}^{-1}$. (a) Temperature distribution, (b) air velocity distribution and (c) flow pathlines from air supply inlets.

Figure 8. MERS-CoV pathways for six source locations with $ach = 6\, \text{h}^{-1}$. (a) Man 1, (b) Man 2, (c) Man 3, (d) Man 4, (e) Man 5 and (f) Man 6.
(with respect to air suspension) and deposited on ward room surfaces, respectively

\[ r_c = \frac{n_c}{n_s}; \quad r_d = \frac{n_d}{n_s}; \quad r_a = \frac{n_a}{n_s} \tag{7} \]

Figure 6 graphs the ratios \( r_c, r_d \) and \( r_a \) against time. The elapsed time \( t_{e_p} \) 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).

\[ r_a(t_{e_p}) = 0 \tag{8} \]

### Results and discussion

#### Airflow field

Figure 7 shows the simulated temperature, air velocity distribution and the flow pathlines inside the ward with \( ach = 6 \text{ h}^{-1} \). 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

Figure 9. Deposited and exhausted ratios of MERS-CoV for six source locations. (a) Man 1, (b) Man 2, (c) Man 3, (d) Man 4, (e) Man 5 and (f) Man 6.
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 Figure 8(c) and (e) show the virus moved along floor surface of the ward but in cases shown in Figure 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 $t_e$ 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 re-suspended particles. Besides, higher $r_e$ and shorter $t_e$ were found with increasing air change rates. If $ach$ was increased from 3 to 13 h$^{-1}$, $t_e$ 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 viruses through better ventilation design strategies. 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.

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

x-axis: Time (s)
y-axis: Deposited ratio $r_d$, exhausted ratio $r_e$
Figure 11. SARS-CoV removal processes for different source locations and air change rates.

x-axis: Time (s)
y-axis: Deposited ratio $r_d$, exhausted ratio $r_e$
Figure 12. H1N1 influenza virus removal processes for different source locations and air change rates.

- x-axis: Time (s)
- y-axis: Deposited ratio $r_d$, exhausted ratio $r_e$
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.

**Author’s contribution**

All authors contributed equally in the preparation of this manuscript.

**Declaration of conflicting interests**

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