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Inter-flat airflow and airborne disease transmission in high-rise residential buildings

Key Messages

1. A virus-spread mechanism is related to inter-flat or inter-zonal airflow through open windows caused by buoyancy effects.
2. Both on-site measurements and numerical simulations quantify the amount of the exhaust air that exits the upper part of the window of a floor and re-enters the lower part of the open window of the immediately upper floor.
3. Ventilation air could contain up to 7% (in terms of mass fraction) of the exhaust air from the lower floor.
4. In high-rise buildings, windows flush with the facade are a major route for the vertical spread of pathogen-containing aerosols, especially those <1 μm in diameter.

Introduction

The outbreak of the severe acute respiratory syndrome (SARS) in 2003 called for closer investigation of the possible transmission routes of infectious diseases in the environment. There were SARS clusters in several high-rise residential blocks in Hong Kong,[1] at Koway Court (Chai Wan) on 25 March 2003, Hing Tung House (Tung Tau (II) Estate, Kowloon City) on 2 April 2003, and Wing Shui House (Lek Yuen Estate, Shatin) on 7 May 2003. Most of the affected households were located along the same vertical block on different floors. The possibility of spread via unsealed U-traps of the drainage systems was ruled out. Especially in the case of Wing Shui House, SARS coronavirus was found in swab samples collected from the wall, windows, and the floors of two units near the index unit, while the residents of these two units had no symptoms of the disease.[1] The vertical spread of the disease suggested that it had been transmitted via airflow under certain environmental conditions.

This study aimed to confirm whether the inter-flat airflow can form a significant path for residents to become exposed to virus-containing aerosols generated by an index patient via sneezing, coughing, and speaking in the flat below. Airflow through open windows was focused, particularly in Wing Shui Building, Shatin, which had single-sided natural ventilation.

Methods

This study was conducted from 1 December 2005 to 30 April 2007. The airborne disease transmission path was investigated using on-site ventilation and tracer gas measurements, as well as computer simulations of the airflow and residual droplet movements.

For on-site measurements of cross contamination of ventilation, three buildings were selected. In each building, two vertically adjacent flats were rented for about one month for tracer gas and ventilation measurements. Two tracer gases SF_6 and CO_2 were used simultaneously to examine contaminant dispersion and ventilation rates of the two selected rooms. SF_6 was used both as a tracer of indoor pollutants originating from the lower floor and for the calculation of air change rate in the lower floor room, and CO_2 merely as tracer for determining the ventilation rates. The SF6 concentrations at six different points within the two rooms were continuously monitored (Fig).

A computational fluid dynamics program solves the governing equations of airflow field in a finite-volume procedure with a staggered grid system. The turbulent effect of flows is simulated by the re-normalisation group k-ε model. The dispersion of gaseous pollutants can be simulated by solving the governing equation of species in air. Since human-generated aerosols mainly range from 1 to 1000 μm in diameter, a relatively large discrepancy may appear for coarse particles whose gravitational settling effect is remarkable. In order to look into the movement of the pathogen-containing droplets, the cascade transport of pollutants in the form of particles was simulated by both Eulerian and Lagrangian methods, as validated in our earlier publication.[2]

Hong Kong Med J 2012;18(Suppl 2):S39-41

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RFCID project number: 03040642

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Results and discussion

The mean SF_6 concentrations of the six monitored points were arranged in descending order as 1.31×10^5, 5.94×10^4, 4.88×10^4, 2.83×10^3, 2.05×10^3, and 1.37×10^3 μg m^{-3}, at points P-3, P-2, P-1, P-4, P-5, and P-6, respectively (Fig). The highest concentration was found at P-3, which represented the source point of the index room. The concentrations at P-1 and P-2, close to the source point, were of the same order of magnitude, with P-2 being slightly higher than P-1. This was related to the route of the airflow. P-2 was at the upper part of the window, which was the outlet of the infected airflow due to buoyancy forces. Fresh air came into the room via P-1, which mixed with the contaminated air, and exhausted through P-2. The concentrations in the upper room at P-4, P-5, and P-6 were much lower than those in the source room at P-1, P-2, and P-3. This indicated that the consecutive dilutions took place along the airflow path during the transmission.

To further quantify this potential inter-flat airflow, two indices were defined based upon a three-zone airflow and a mass balance model. The mass fraction M_{i-j} was defined as the mass fraction of air that originated from the point i in the lower source room and was present in the point j of the upper room. By assuming a quasi-steady airflow process, the mass fraction M_{i-j} can be directly calculated from the monitored tracer gas concentrations using equation 1:

$$M_{i-j} = \frac{Cj}{Ci},$$

where Ci and Cj were the monitored tracer gas concentrations in the source room and the secondary room. Another index, the re-entry ratio k, was defined as the fraction of the exhaust air from the lower source room which re-entered the adjacent/upper room. By assuming a steady state airflow and mixing process and well mixed conditions for both rooms, the re-entry ratio k can be calculated using equation 2:

$$k = M_{i-j}\ \frac{V_2(ACH)_2}{V_1(ACH)_1},$$

where V_1 and V_2 were the room volumes and $(ACH)_1$ and $(ACH)_2$ the measured air change rates (hr^{-1}).

As far as the spread of infectious disease is concerned, the index mass fraction M_{i-j} may be the more direct index. As an extension of the well-mixed assumption, two local mass fractions, M_{2-4} (the fraction of air present at P-4 and originating from P-2) and M_{2-6} (the fraction of air present P-6 and originating from P-2) were calculated from equation 1 (Table 1). The value of M_{2-4} was larger than M_{2-6}, as P-6 was further away from the source. Hence, P-6 had more dilution than P-4, as described in the previous section. The maximum mass fraction M_{2-4} was about 0.07, which meant that the air near the window upstairs contained 7% of the exhaust air originating from the lower room. The current ASHRAE ventilation standard[3] promulgates that the dilution factor should exceed the value 50 to 1 when highly hazardous pollutants are involved, which would correspond to the mass fraction round 0.02.[3] Increasing wind speed was shown to suppress the value of the mass fraction. At a low wind speed of 0.03 m/s, the mass fractions at the two points in the upper room reached to values of M_{2-4}=0.07, M_{2-6}=0.029, respectively. When the wind speed was at the higher end (0.87 m/s), the mass fractions were reduced to M_{2-4}=0.04, M_{2-6}=0.016. At both low wind and small indoor-outdoor temperature differences, the mass fraction tended to be higher, which agreed with our hypothesis that pure but low buoyancy driven flow on calm days may pose the highest infection risks.

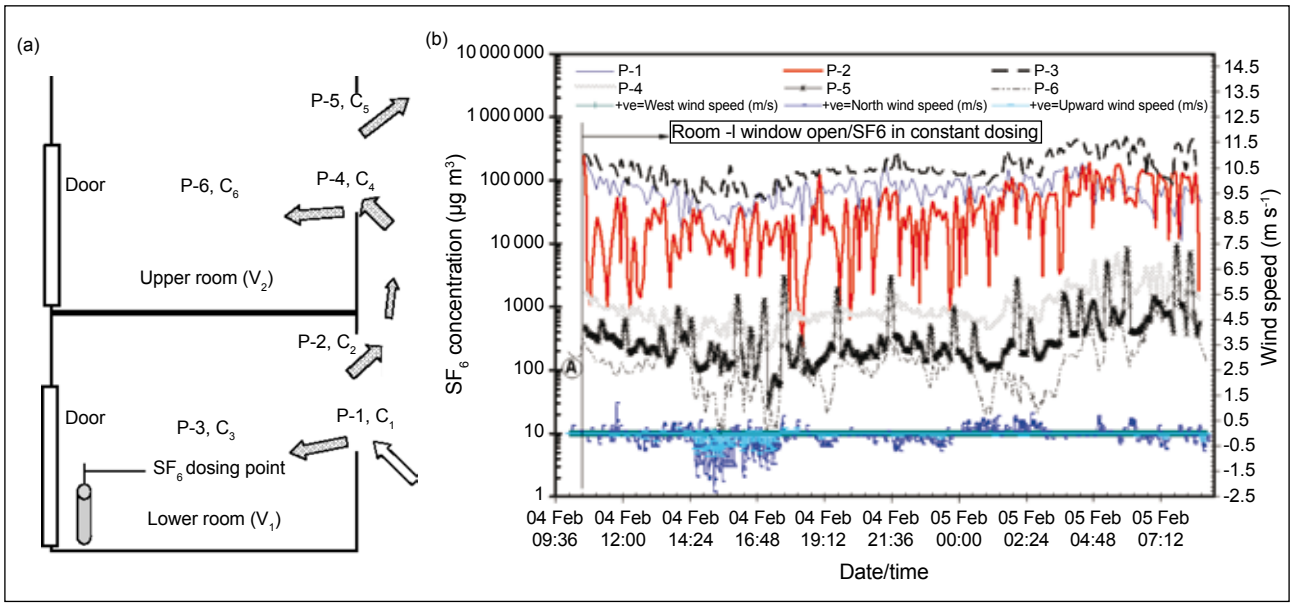

Fig. (a) Tracer-gas dosing and sampling points in the two adjacent floors upstairs and downstairs. (b) Example concentration profiles of tracer gas SF_6 at the six points on two low-wind days

Table 1. Mass (M) fractions, ratio of air change rates per hour (ACH) of the two rooms, re-entry ratio of exhaust air at different wind ranges

Wind speed (m/s)	M_{2-4}	M_{2-6}	$(ACH_1)/(ACH_2)$	Re-entry ratio *k*	No. of data set
≤0.03	0.070	0.029	1.64	0.048	4740
0.25	0.055	0.026	1.71	0.044	2080
0.45	0.050	0.022	1.73	0.038	1306
0.66	0.046	0.019	1.74	0.033	653
0.87	0.041	0.016	1.75	0.028	270
1.07	0.039	0.011	1.69	0.019	135
1.3	0.038	0.006	1.66	0.01	82
1.5	0.032	0.007	1.65	0.011	46
1.69	0.035	0.005	1.67	0.009	43
1.88	0.038	0.005	1.61	0.009	32
2.11	0.033	0.005	1.64	0.008	19
2.48	0.036	0.004	1.75	0.006	11

Numerical simulations

Based on the knowledge of infection dose (the number of organisms required to cause infection), the risk of airborne infection and ventilation rate per person can be correlated by the Wells-Riley equation.[4] Assume that one patient standing at the middle of the second floor producing 13 infectious quanta per hour, pulmonary ventilation rate to be 0.6 m^3/h, and exposure time of 8 hours, the calculated mean infection probabilities can be as high as 6.6% on the third floor (Table 2).

The upper to lower concentration ratio for 1.0 μm particles was close to the values for CO_2. The results of 10.0 and 20.0 μm particles differed from the 1.0 μm particle remarkably. Different from gaseous pollutants, two factors constrained the cascade effect of coarse particles: (1) the effect of gravity on the particles which counteracts the upward buoyancy force, and (2) the absorption of particles on solid surfaces. The latter is a self-cleaning mechanism of particles, which contributes to decreasing suspended concentrations in the lower floor and reducing outlet concentrations.

Conclusions

On-site airflow visualisation, tracer gas measurements, and numerical simulations revealed qualitatively and quantitatively the vertical upward re-entry possibilities of the exhaust air in high-rise residential buildings by open window ventilation practices. The presence of exhaust air from a lower room in an adjacent upper room can reach up to 7%, in terms of mass fraction. With regard to the transmission risk from a lower floor to the floor immediately above via this route, the order is one magnitude lower than the risk in the same household. But this risk should not be overlooked. As one of the effective intervention measures is to isolate and quarantine the close-contacts, the upstairs household residents may be included in the close-contact list in the event of a highly infectious disease.

Table 2. Mean risk of infection calculated from the Wells-Riley equation (based upon 8-hour exposure, using *Mycobacterium tuberculosis* as the pathogen)

Mean risk of infection on	Infection probability at wind speed of				
	0.1 m/s	0.5 m/s	1.0 m/s	2.0 m/s	4.0 m/s
Second floor	30%	28%	29%	31%	46%
Third floor	2.0%	3.4%	3.5%	6.6%	1.7%

Acknowledgement

The study was supported by the Research Fund for Infectious Disease Control, Food and Health Bureau, Hong Kong SAR Government (#03040642).

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