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Role of air distribution in SARS transmission during the largest nosocomial outbreak in Hong Kong

Abstract Severe acute respiratory syndrome (SARS) is primarily transmitted by bio-aerosol droplets or direct personal contacts. This paper presents a detailed study of environmental evidence of possible airborne transmission in a hospital ward during the largest nosocomial SARS outbreak in Hong Kong in March 2003. Retrospective on-site inspections and measurements of the ventilation design and air distribution system were carried out on July 17, 2003. Limited on-site measurements of bio-aerosol dispersion were also carried out on July 22. Computational fluid dynamics simulations were performed to analyze the bio-aerosol dispersion in the hospital ward. We attempted to predict the air distribution during the time of measurement in July 2003 and the time of exposure in March 2003. The predicted bio-aerosol concentration distribution in the ward seemed to agree fairly well with the spatial infection pattern of SARS cases. Possible improvement to air distribution in the hospital ward was also considered.

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Key words: Severe Acute Respiratory Syndrome; Infection Control; Air Distribution; Computational Fluid Dynamics; Hospital Ventilation; Airborne Transmission.

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Practical Implications
Our study revealed the need for the development of improved ventilation and air-conditioning systems in an isolation ward or a general hospital ward for infectious respiratory diseases. The outbreak in Ward 8A, which was in a general hospital and could house nearly 40 patients, demonstrated the cross-infection risks of respiratory infectious diseases in hospitals if a potential highly infectious patient was not identified and isolated. Our example simulation, which extended the SARS Busters' design for an isolation room to Ward 8A, demonstrated that there was room for improvement to minimize cross-infection in large general hospital wards.

Introduction

As a new human disease, the epidemic of severe acute respiratory syndrome (SARS) between November 2002 and June 2003 resulted in unprecedented international efforts to control the disease. The epidemics caused a significant impact on regional economy and health care systems. SARS originated in Guangdong, China, and spread to the rest of the world when an infected medical doctor from Guangdong stayed in 'Hotel M' in Hong Kong in late February 2003, where the disease subsequently infected at least 14 other guests and visitors to the hotel (Anonymous, 2003). Some of these infected individuals sparked large outbreaks in hospitals in their own countries/cities. Hong Kong had a total of 1755 cases and a death toll of 299 [World Health Organization (WHO), 2003a].

A 26-year-old Hong Kong resident visited Hotel M in late February during the Guangdong doctor's stay, and contracted the infection. He was treated in Ward 8A at the Prince of Wales Hospital, which subsequently led to a large SARS outbreak (Lee et al., 2003) from March 11 to 25, 2003, with 138 probable cases. The patients included 69 health care workers, 16 medical students who were attending clinical teaching or examinations in the ward, and 53 patients/visitors

who were either in the same ward or had visited their relatives there. Detailed epidemiological studies on the spread of SARS among the three different groups of victims have been reported elsewhere [see Lee et al. (2003) for health care workers; Wong et al. (2004) for medical students; and Yu et al. (in press) for inpatients].

The SARS virus was mostly spread by close personal contact and large droplet transmission (WHO, 2003b). Possible airborne transmission was documented for some super-spreading events in the SARS epidemics, for example, in a hospital outbreak in Canada (Christian et al., 2004) and the Amoy Gardens outbreak in Hong Kong (Yu et al., 2004). An understanding of the possible causes of airborne transmission is crucial for developing and selecting appropriate and effective engineering control methods in both hospitals and the community.

It was known that many other respiratory viruses, such as those causing the common cold and flu, could spread from an infected person to a new host by airborne bio-aerosol inhalation, personal contact such as handshaking, and by touching contaminated surfaces. Past studies on transmission routes for communicable respiratory infection were reviewed by Barker et al. (2001) for community facilities and domestic homes, by Mendell et al. (2002) for work places such as offices, and by Cole and Cook (1998) for health care facilities. Most studies on airborne transmission of various respiratory viruses were not as conclusive as for the person–person, or person–surface–person transmissions. A study by Duguid (1945) showed that sneezing and coughing could generate a million or so droplets up to 100 μm in diameter plus several thousand larger particles, while a recent study by Papineni and Rosenthal (1997) showed that for healthy individuals, the number of droplets generated during respiratory activities was much less. As droplets were humid, they started to evaporate after release and thus change their mass and size. Their size could be sufficiently small (0.5–12 μm) to be airborne (Cole and Cook, 1998). This also meant that if the large particles originally settled because of the effects of gravity, they could be resuspended as they evaporated and became smaller. The settling velocities for these small particles (0.5–12 μm) were very low at between 0.05 and 0.3 mm/s. The rate of evaporation was dependent upon the ambient humidity. As the indoor relative humidity was generally controlled to be 50–60% in an air-conditioned room, the sizes of droplet in diameter of less than 100 μm reduced rapidly once released into the air; see Wells (1934) and Brundrett (1992). The airborne infectious particles were often considered to be droplet nuclei. Rudnick and Milton (2003) used CO_2 as a marker for estimating the risk of indoor airborne infection.

To assist the epidemiological studies of the Ward 8A outbreak carried out by Wong et al. (2004) and Yu et al. (in press), we carried out ventilation and air distribution studies in Ward 8A. Retrospective on-site inspections and measurements of the ventilation and air distribution system were carried out in July 17, 3 months after the outbreak. Limited on-site measurement of bio-aerosol dispersion was carried out in Ward 8A on July 22. Computational fluid dynamic (CFD) simulations were performed to analyze the bio-aerosol dispersion in the hospital ward. We attempted to predict the airflow distribution in Ward 8A at the time of exposure in March 2003 and at the time of measurement in July 2003. The latter provided an opportunity to evaluate our predictions and also to study the effects on bio-aerosol dispersion of the number of beds in the ward. Possible improvements to air distribution in the hospital ward were also considered. This paper presents the detailed results of this ventilation study. We discuss the association between the spatial infection pattern and the predicted bio-aerosol dispersion pattern. The association to be revealed later clearly demonstrates the significance of air distribution control in hospital wards.

Infection distribution patterns in Ward 8A

Ventilation design in Ward 8A

The floor plan of Ward 8A at the time of the SARS outbreak in March 2003 is shown in Figure 1. The ward had four main cubicles, separated by a corridor and a nurses' station, a store and a store/cleaning room. The cubicles were semi-enclosed. There were normally eight beds in each of the four cubicles, but at the time of the outbreak 10 beds were in each cubicle. The ward had an overall dimension of 24 m (length) $\times$ 18 m (wide) $\times$ 2.7 m (high). Each cubicle was $7.5 \times 6 \times 2.7$ m. Ward 8A was centrally air-conditioned. Fresh air was drawn from outside the hospital building into a primary air unit situated in a room adjacent to the ward entrance, where it was cooled by chilled water and then supplied to this ward (and another ward on the opposite side of the hospital) through air ducts. The air was then distributed to five fan-coil units (one in each of the four cubicles and one over the nurses' station), where it was mixed with recirculated air, cooled by chilled water, and blown into the cubicle/nurses' station via four-way air supply diffusers (0.6×0.6 m) located at the center of the cubicle in the false ceiling and over the nurses' station. An exhaust grille (0.3×0.6 m) located in the false ceiling in the corridor outside each cubicle and outside the nurses' station, recirculated 70% of the air supply back into the fan-coil unit. Excess air escaped through two extraction fans inside the patients' toilet, two extraction fans in the store/cleaning room, and through

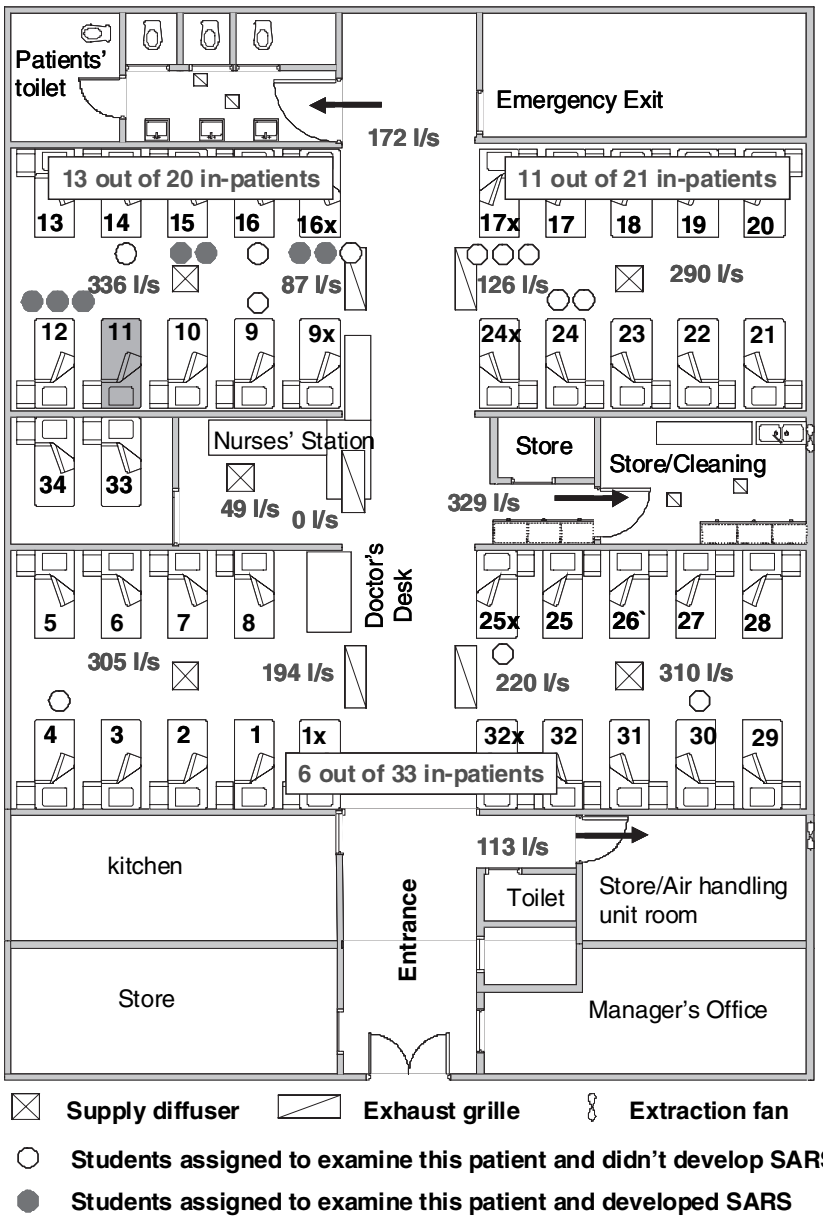

Fig. 1 Floor plan of Ward 8A during the time of outbreak in March 2003. There were four large cubicles, each with 10 beds. Measured supply and exhaust flow rates (in liter per second, or l/s as shown here) are shown for each diffuser/grille. The bed (no. 11) where the index patient stayed is also marked. The spatial distribution of the infected medical students and inpatients is also shown. The location of the 19 (out of 20) medical students who attended the 40-min bedside clinical assessments on March 6 and 7 are shown by bullets (developed SARS) and circles (did not develop SARS). The numbers of inpatients who developed SARS in each cubicle are also marked [prepared using data and figures from Wong et al. (2004)].

the ward to the entrance and then to an extraction fan in the air-handling unit (AHU) room.

The air change rate was measured to be 7.8 air changes per hour for the whole ward. The measured supply and exhaust airflow rates on July 17 are marked in Figure 1. At the time of measurement, additional extraction fans were installed in the windows in all four cubicles. These extraction fans were turned off during the measurement period. We did not measure the infiltration/exfiltration rates through the building envelope leakage in the ward, but because of the openings in the extraction fans, the infiltration rates might have affected the bio-aerosol dispersion in the ward at the time of measurement. Because of the large number of air change per hour (7.8), the infiltration/exfiltration through wall/door/window leakage areas would not have significantly affected the airflow pattern and bio-aerosol dispersion pattern in the ward. The airflow pattern was dominated by the mechanical supply and extraction system. We were also not able to measure the extraction flow rate through the extraction fan in the AHU room. This flow rate was obtained by

assuming that the total inflow and outflow rates were balanced in the ward.

The supply and exhaust airflow rates through the supply diffusers and exhaust grilles in the ward were found to be unbalanced, as shown in Figure 1. The air supply from the diffuser in the index patient's cubicle had the highest supply flow rate (336 l/s), while the adjacent exhaust grille had the lowest exhaust flow rate (87 l/s) among all four functional exhaust grilles. The exhaust and air supply for the nursing station did not function properly. The unbalanced air supply and exhaust among all diffusers/grilles could also have affected the airflow pattern and bio-aerosol dispersion in the ward, or between cubicles which will be shown later.

Ward 8A spatial infection pattern – medical students

Wong et al. (2004) provided the epidemiological features and patterns of transmission of SARS among the medical students. In summary, the index patient was admitted to Ward 8A on March 4 and was placed in Bed 11. His cough persisted from March 4 to 13, and was most severe from March 4 to 7. A jet nebulizer was used to treat him four times per day starting from 2 pm on March 6 until March 12, with each treatment lasting about 30 min. The use of nebulizers was initially suspected of contributing to the SARS virus transmission in the Ward 8A outbreak (Lee et al., 2003), and subsequently banned for use in Hong Kong hospitals in SARS patients. However, Wong et al. (2004) found that among all the students studied, no significant association was noted between their risk of illness and their presence in the ward when the nebulizer was in use. Thus, in this study, we assumed that the main virus source was because of coughing of the index patient.

On March 12, the 26-year-old man was identified as the index patient for the outbreak of SARS in the Prince of Wales Hospital, and he was transferred to the isolation room within the ward where Beds 34 and 33 were located (Figure 1).

It was difficult to analyze the infection data on health care workers with regard to the possible existence of a spatial pattern, because doctors and nurses moved around in the ward, with highly possible close contacts with the index patient. It was also difficult to analyze the infection data on visitors as it was very difficult to find the denominator to estimate the attack rate at different bed locations. However, the other two groups, i.e. the medical students and the patients, provided useful information on spatial infection patterns.

The group of 20 third-year medical students who visited the ward had well-defined exposures. The students performed bedside clinical assessments in the ward on the mornings of March 6 and 7 (see Figure 1). Each student examined specific patients in the ward during a 40-min period on either March 6 or 7. The locations (bed numbers) of the patients assigned to each student were known and are shown in Figure 1. None of the 20 students who visited this ward had any contact with other SARS patients elsewhere after March 7. In addition to the students who performed the bedside assessments, 46 other students (mostly fifth-year students) also visited the ward for bedside teaching or clinical training during March 4–10.

Sixteen (24%) of the 66 medical students subsequently developed SARS. The dates of the onset of illness of the medical students are shown in Figure 2. If the virus-laden bio-aerosols or droplets were uniformly distributed in the ward, there would have been a uniform distribution of infection risk in the ward. Wong et al. (2004) calculated the attack rates of the illness among students, based on whether the students could recall entering the index patient's cubicle. SARS developed in 10 of the 27 students who reported entering this cubicle, compared with four of the 18 students who could not accurately recall whether they entered the patient's cubicle, and in only one of 20 students who reported that they never entered the cubicle. This epidemiological result clearly suggested that the virus-laden bio-aerosols were not uniformly distributed throughout the entire ward, presuming the transmission of the disease was airborne.

The epidemiological study also showed that there was a clear association between the proximity of exposure and the infection. Among 19 of 20 medical students (excluding the ill student who had an unusually long incubation period) who attended the bedside clinical assessments on March 6 or 7, seven developed SARS (see Figure 1). All three students who examined patients located in beds within 1 m of the index patient developed SARS; four of eight students in the same cubicle but in beds >1 m from the index patient developed SARS; but none of the eight students in other cubicles fell ill. This particular spatial infection pattern could not be explained by a single transmission route, such as large bio-aerosol droplets or close personal contact. Large droplet transmission sneeze or cough was normally considered to be effective within 1 m of the index patient; see Centers for Disease Control and Prevention (2003, p. 6).

Ward 8A spatial infection pattern – inpatients

The SARS virus transmission among the inpatients in this outbreak was studied. A total of 74 inpatients stayed in the same ward as the index patient during the period March 4–12. They were allocated to specific beds during their stay and were not as mobile as the health care workers. Visiting the toilet and passing through the corridor area might also have led to inhalation of virus-containing bio-aerosols or exposure through surface contact, in which case the

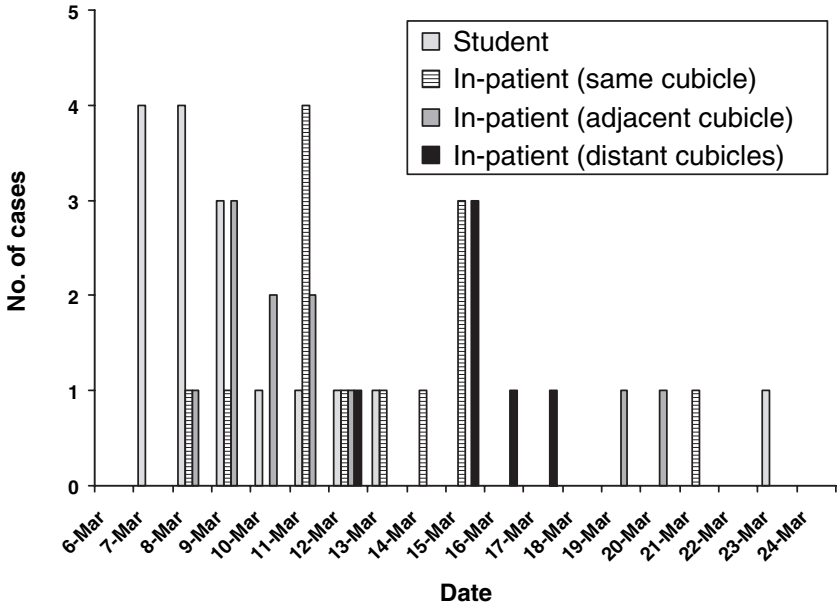

Fig. 2. Date of onset of illness of medical students and inpatients in different cubicles with severe acute respiratory syndrome [prepared using data from Wong et al. (2004)].

spatial infection pattern should have been rather uniform throughout the ward, but that was not the case.

When the index patient was admitted on March 4, there were 36 patients in Ward 8A. In the consecutive days after March 4, 7, 7, 8, 2 and 14 patients were admitted, with no more being admitted after March 9. Of these patients, 30 developed SARS during the follow-up period. There seemed to be an association between the date of stay in the ward and SARS infection. Of the 41 patients who were present in the ward on March 6, 23 developed SARS, while only seven of the 33 patients who were not present on March 6 did. None of the nine patients discharged from the ward before March 6 developed SARS. This probably indicated that a significant number of infected inpatients might have inhaled (or acquired) a sufficient dose of the SARS virus on March 6. However, it was not possible to identify the exact time slot when the infection actually occurred. It was interesting to note that among the 20 medical students who attended bedside clinical examinations, those who attended the March 6 session had a higher attack rate (Wong et al., 2004). It was known that the March 6 and 7 sessions were held in the morning (10:00 am to 12:40 pm). Each assessment lasted about 40 min. We might suspect that the morning of March 6 might have been the high-risk period for infection in this outbreak; however, it was still not possible to further narrow down the period of time when the infection occurred. It was also highly likely that the infection had occurred at different times during March 4–12. The existence of multiple peaks in the epidemiological graph shown in Figure 2 indeed showed that there were possibly multiple infection periods. The uncertainty in pinpointing the exact infection time had presented difficulties in our airflow pattern study. Various physical parameters affected airflow and bio-aerosol dispersion in the ward, such as heat gains through the ward envelope, solar heat gains through windows and supply air temperatures, which were all a function of time.

There was clearly a spatial infection pattern among the infected inpatients (see Figure 1). Thirteen of the 20 inpatients staying in the same cubicle as the index patient were infected with SARS (an attack rate of 65.0%), while 11 of 21 inpatients in the adjacent cubicle (i.e. the top-right cubicle in Figure 2) developed SARS (attack rate 52.4%), and only six of the 33 inpatients in the two distant cubicles (i.e. bottom two cubicles in Figure 2) fell ill (attack rate 18.2%).

The studies by Wong et al. (2004) on medical students on inpatients showed that airborne spreading of the SARS virus could not be ruled out in the Ward 8A outbreak. There seemed to be an association between the spatial infection pattern and the dispersion of virus-containing bio-aerosols originating from the index patient. Thus, there is a need for a detailed study of the air distribution design in Ward 8A at the time of exposure, not only to provide further detailed environmental evidence of airborne transmission, but also to identify or develop appropriate engineering control systems. The results may be very useful in improving ventilation design and developing new air distribution methods for hospital wards.

Methodologies for airflow prediction

Predicting airflow patterns at the time of outbreak

Some modifications were carried out to improve the ventilation system in Ward 8A soon after the March SARS outbreak. Extraction fans were installed in windows in each cubicle to create a negative pressure environment. The number of beds in each cubicle was reduced from 10 to 8. Thus, at the time the measurements were carried out in mid-July 2003, some of the parameters affecting airflow patterns in the ward had already been changed.

We considered the following methodologies for investigation. We first performed a simple measurement in the ward in mid-July when there were eight beds in each cubicle. The supply and exhaust airflow rates were measured by a hood flow rate meter (APM 150; TSI Inc., Shoreview, MN, USA) (measurement range 24–945 l/s with an accuracy of 3%). Air velocity, air temperature and relative humidity at all supply diffusers and exhaust grilles were measured by a portable Velocicalc Plus air velocity meter Model 8386A (TSI Inc.). The available time for measurement was limited to 4 h on each of two afternoons, as the ward was still occupied by (non-SARS) patients. The exhaust fans were turned off during the time of measurement, which was possible as there were no SARS patients in the ward and so the negative pressure environment was not required. A floor plan showing the location of all beds at the time of exposure was obtained from the hospital. Information on the ward ventilation system was also obtained from the Electrical and Mechanical Services Department of the hospital. No flow balancing was carried out in the ward between March and July 2003. Thus, we assumed that the supply and exhaust airflow rates through each diffuser/grille had not been changed since the outbreak when the on-site measurements were undertaken on July 17 and 22. On-site measurements also allowed us to obtain all necessary information on room sizes, airflow rates through supply diffusers, exhaust grilles and extraction fans in the ward.

We then carried out CFD simulations to analyze the airflow patterns and the dispersion of virus-laden bio-aerosols at the time of measurement, with a hypothetical index patient in the bed close to the original index patient's bed. The analyzed results were compared with the measurements. This process allowed us to establish some of the key parameters, as well as to determine the appropriate computational domain for further analysis.

The airflow patterns at the time of exposure were then predicted using CFD simulations. Our predicted bio-aerosol dispersion pattern was compared to the spatial infection pattern in the ward for both medical students and patients. Additional CFD simulations were carried out to study what would have happened if there were only eight beds in each cubicle at the time of exposure; and the effects of supply airflow uniformity and improved ventilation design. Both the measurements and the CFD simulations are for steady state conditions.

CFD simulations

Our computational domain included all four cubicles and the corridor, as well as the entrance. The toilets, store, kitchen and the manager's office were excluded in the computational domain. The isolation cubicle had an independent ventilation system and its door was normally closed. The isolation cubicle was excluded from our computational domain.

Each supply diffuser (a square four-way diffuser) was divided into four equal sections, and each section was modeled as an opening. The airflow radiated out at a direction of 30° from the horizontal ceiling. The supply airflow rate was divided equally among the four sections. The measured supply airflow rate and supply air temperature in each diffuser were used as the boundary conditions. As a reference, the mean supply air temperature was 14.3°C. The measured airflow rates at each exhaust grille were also used as boundary conditions. The doors of the patients' toilet, the AHU room and the store/cleaning room were assumed to be half open, with a specified exhaust flow rate as measured from the extraction fans. It was difficult to estimate the heat loss from the external walls, as the exact hour when the infection occurred in early March was unknown. The convection and conduction heat gain/loss through the external wall was not considered, neither was the radiation between interior surfaces. A total heat gain of 11.64 kW, including 2.232 kW because of lighting, 2.964 kW because of 39 patients (76 W each and 50 W/m^2), and 6.444 kW uniformly distributed on the floor, was used for both the time of exposure in March and the time of measurement in July. Each patient was modeled as a rectangular prism. The virus-containing bio-aerosols were modeled as a passive tracer (CO_2). It was found that the water droplets evaporated rapidly after release, which justified the use of a passive tracer in our calculations (Wells, 1934). The virus-containing bio-aerosols originated from the respiratory activities of the index patient. We also neglected the initial exhalation velocities. A small volume of air above the index patient was used to specify the passive tracer source. The source term of CO_2 was estimated from the exhalation flow rate. Only steady-state conditions were considered.

The commercial CFD software Fluent 6.1 (Fluent USA, Lebanon, NH, USA) was used. In the unstructured finite volume method, we chose to use a second-order upwind scheme for all convection terms. The RNG turbulence model was chosen because of its

relatively good accuracy in modeling indoor airflows, as shown by previous studies (e.g. Chen, 1995). For each simulation, about one million grid points were used. The results were then analyzed using the independent graphics package Tecplot9.2 (Tecplot, Inc., Bellevue, WA, USA).

Simulation results and analysis

Airflow patterns in Ward 8A

There were eight beds in each cubicle at the time of measurement in July 2003. We found by simulation that the airflow patterns at the time of measurement in July and at the time of exposure in March were very similar. The airflow pattern in the ward was dominated by the radiating supply jets from the ceiling diffusers in the four cubicles (Figures 3 and 4). The cold supply air from a diffuser radiated out in four directions just below the ceiling, establishing four separate supply jets. The spread of the supply jets was greatest for the index patient's cubicle as it has the highest supply airflow rates. The jets facing the end wall penetrated deeper than those facing the corridor in all cubicles, because of the warmer corridor air. The supply jets facing the end wall impinged to the wall and turned down around the corner, which provided sufficient momentum to generate a global flow recirculation in each cubicle [Figures 5 (bottom) and 6 (bottom)]. The end wall jets created lower pressure regions that caused the lateral supply jets to bend toward the walls. The warm air was pushed from each cubicle to the corridor area, where a warm region was established [Figures 5 (top) and 6 (top)]. The relatively high air temperature in the corridor area weakened the supply air jets facing the corridor. The body force of these jets eventually overcame their initial momentum, and the jets fell down and dispersed. These figures do not show the weak thermal plumes that rose above each patient, which interacted with the global air recirculation in each cubicle. Most body plumes in the end wall areas were destroyed.

The air temperature distribution in two vertical planes that cut through the supply diffusers are shown in Figures 5 (top) and 6 (top). The air temperature distribution in the cubicle was of the mixing type. The air temperature in the index patient's ward was slightly lower than the other cubicles, as the supply flow rate in the index patient's cubicle was high. The temperature distribution was similar in all four cubicles. The corridor area was generally warmer (one degree higher) than the cubicles, which might be undesirable, as health care workers mostly used the corridor area. Health care workers generally had a high clo value (relatively high cloth insulation) because of the use of personal protective equipment and a high met value (relatively high metabolic rate) as they moved around the ward. This might justify the addition of a dedicated air supply diffuser in the corridor area for health care workers in a ward like 8A. The warm air accumulated in the corridor region before being moved out through the ceiling exhaust grilles, and through the extraction fans in the toilet and the storeroom.

The airflow pattern in the ward at the time of exposure was predicted to be very similar to that at the time of measurement in July (Figures 3–6). The addition of two beds in each cubicle meant an additional heat power of 148 W (76×2) in each cubicle. The average air temperature in each cubicle would have increased by 0.4°C at steady state. In practice, this

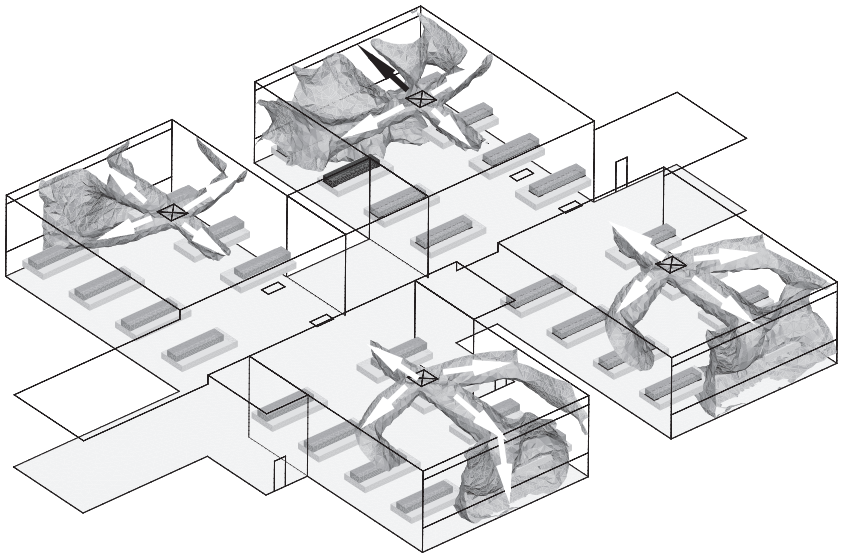

Fig. 3. Iso-surface at 20°C showing the supply airstreams spreading in four individual directions in each cubicle at the time of measurement. The airflow direction is indicated by black/white arrows

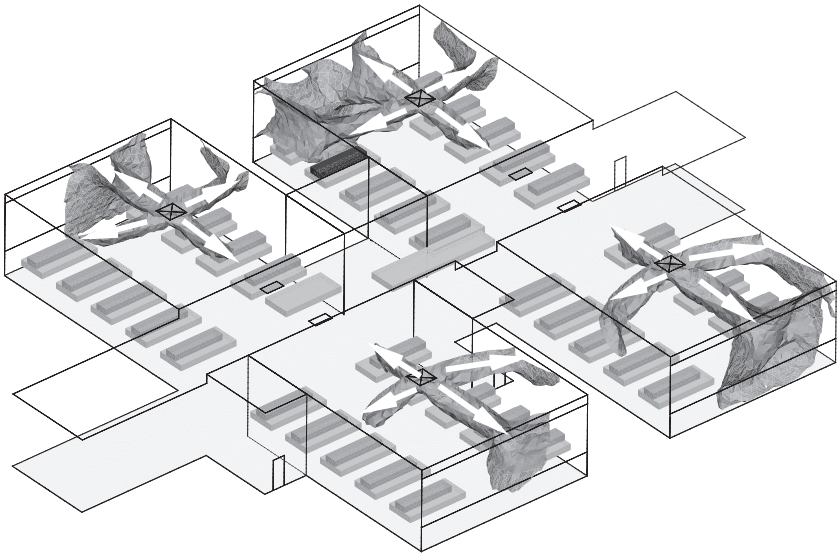

Fig. 4. Iso-surface at 20°C showing the supply airstreams spreading in four individual directions in each cubicle at the time of outbreak. The airflow direction is indicated by white arrows

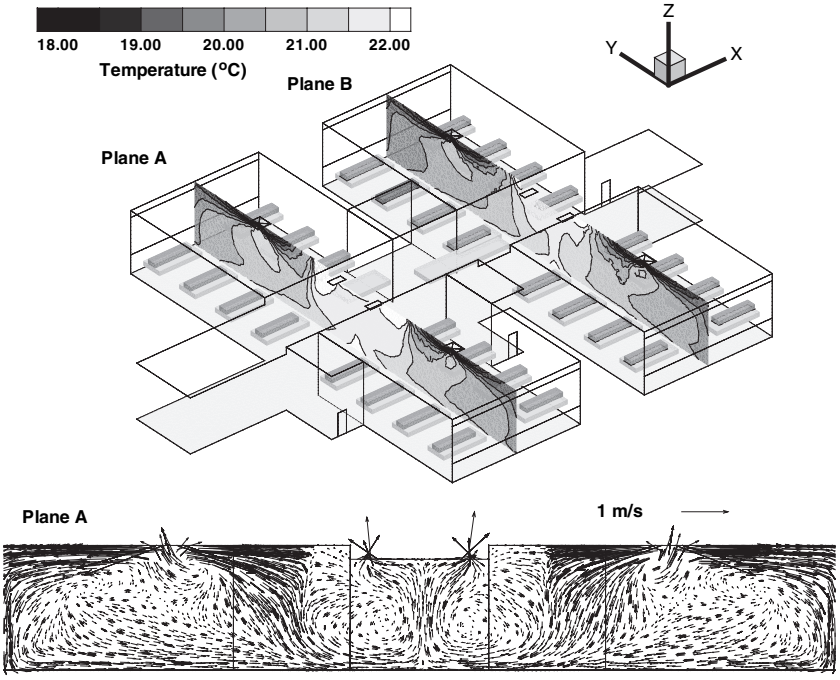

Fig. 5. (Top) Air temperature distribution in two vertical planes in the middle of the cubicles at the time of measurement. (Bottom) Velocity vectors in one of the two vertical planes shown above at the time of measurement

increase of ward air temperature might be automatically controlled by reducing supply air temperatures through the diffusers.

The jet penetration lengths were reduced at the time of exposure (Figure 4) as compared with those at the time of measurement (Figure 3). The airflow patterns found in Ward 8A at the time of measurement and at the time of exposure were common in general medical hospitals in Hong Kong and probably in other countries. During and after the SARS epidemics in Hong Kong, Hong Kong engineers tested a new air-conditioning system for SARS isolation wards with multiple beds (Li and SARS-Busters, 2003; SARS Busters, 2005).

To demonstrate the possible improvements to the ventilation in Ward 8A, we considered the SARS Busters' design in each cubicle in the ward. The original design was not for general medical wards, but for

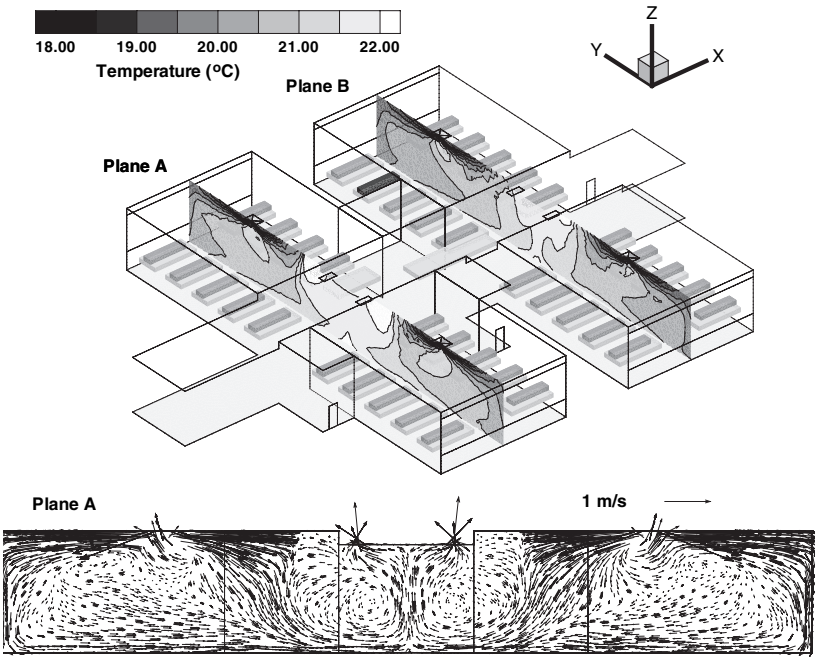

Fig. 6. (Top) Air temperature distribution in two vertical planes in the middle of the cubicles at the time of outbreak. (Bottom) Velocity vectors in one of the two vertical planes shown above at the time of outbreak

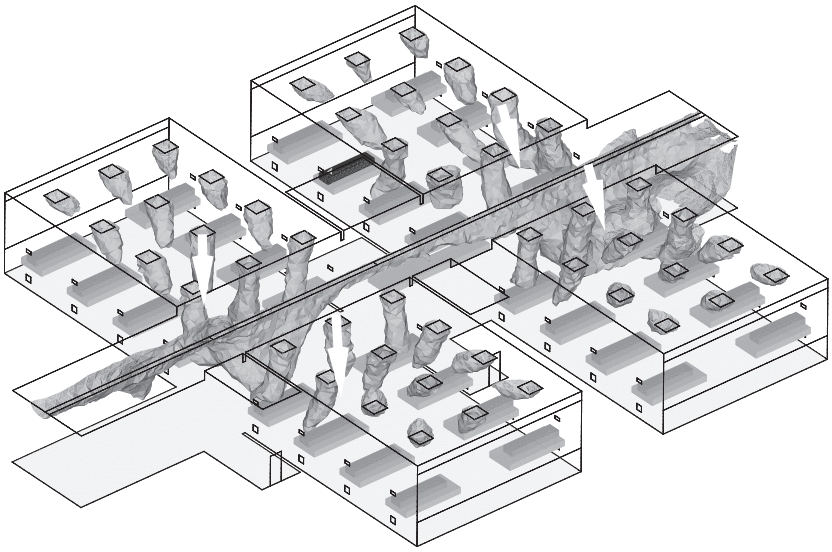

Fig. 7. Iso-surface at 19.2°C showing the supply airstreams spreading in the ward from a total of 48 downward supply grilles and one linear downward supply diffuser in the corridor (SARS Busters'system)

isolation wards with multiple beds. The design included a dedicated downward supply diffuser for each bed, with four supply diffusers over the aisle between the two rows of beds in each cubicle (Figure 7). Additionally, a linear downward diffuser was added in the corridor ceiling to provide an 'air curtain' to minimize the mixing between adjacent cubicles. Two levels of exhaust were designed in each cubicle for each bed – the floor level exhaust and the bed-head level exhaust. The exhaust airflow ratio between the two exhausts was 70:30, with the bed-head level having a smaller flow rate (Li and SARS-Busters, 2003). The extraction flow rates in the patients' toilet, the store/cleaning room and the AHU room were kept the same. Thus, in each ward, there were 12 supply diffusers (0.5×0.5 m) and 16 exhaust outlets. Obviously, this was a difficult ventilation system in practice because of space and financial constraints. We were interested in

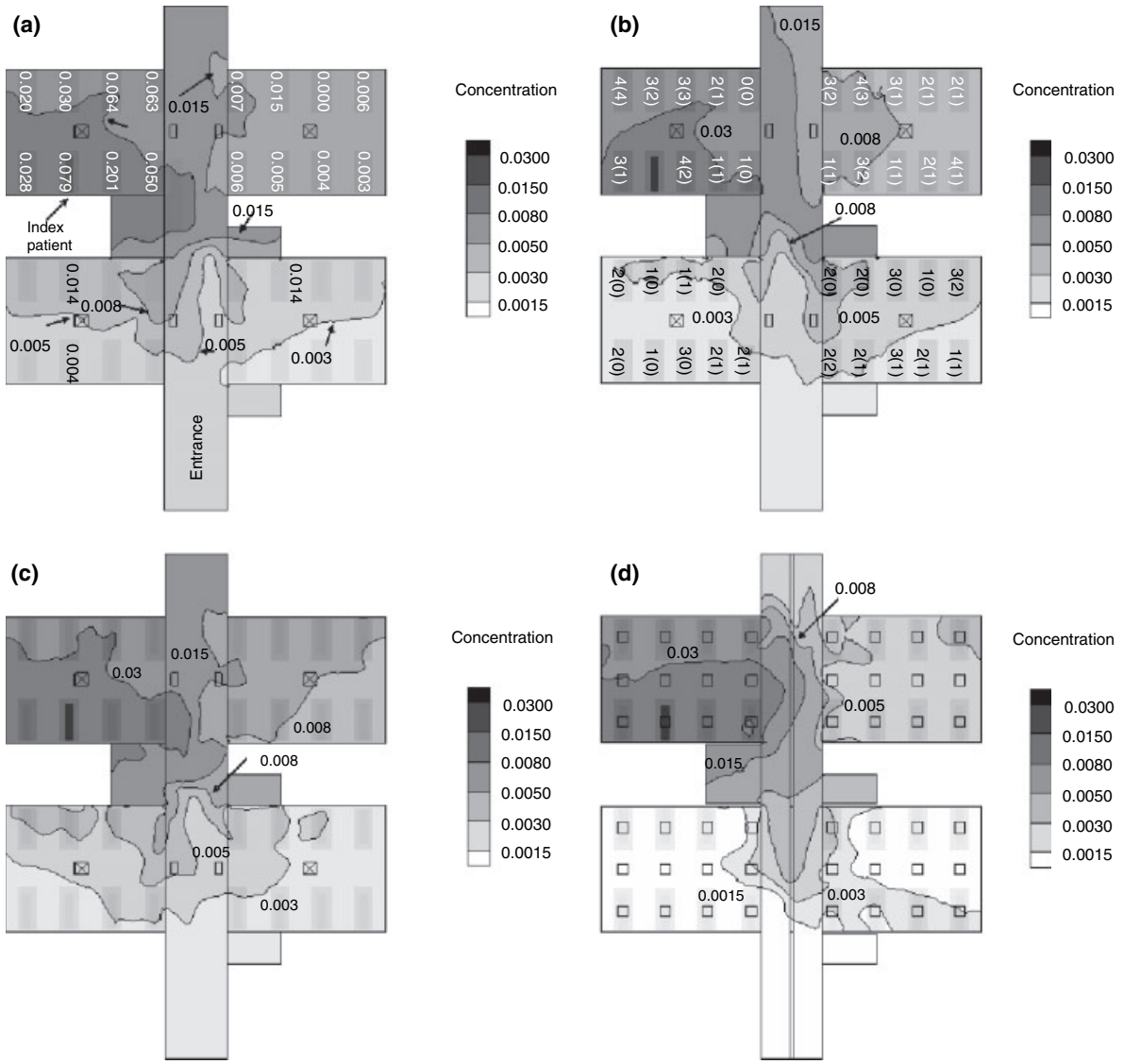

Fig. 8. Distribution of normalized virus-laden bio-aerosol concentrations at a height of 1.1 m in Ward 8A under different conditions. (a) At the time of measurement in July 2003 – measured concentrations in selected beds are also shown. The numbers on the beds are the normalized measured concentrations, with a white color in the two top cubicles and black color in the two bottom cubicles; (b) at the time of outbreak in March 2003 (the total number of inpatients, *X*, who stayed in a bed between March 4 and 12 and the number of those who developed SARS, *Y*, are marked in each bed as *X*(*Y*); (c) at the time of measurement if the supply/exhaust flow rates through all diffusers/grilles were balanced; and (d) at the time of measurement if a downward ventilation system (i.e. the new hypothetical ventilation system) was installed.

exploring the potential to improve ventilation performance.

Figure 7 shows the supply jets and their penetration into the ward. The eight supply diffusers in each cubicle were located just above the foot of each bed to avoid a possibly cold direct downward flow to a patient's head. The supply airstreams were different from one bed to another. For beds close to the end walls where the air temperature was relative high, the supply streams mixed rapidly with the air in the ward. The 'body' temperatures of the 'patients' in these beds were higher than those close to the corridor areas. For beds adjacent to the corridor, downward flows resulted as designed, but there was a risk of a cold draft for patients as some downward flows changed direction. The body temperatures of these patients were found to be relatively low. The downward flows of the linear diffusers in the corridor were not steady, as they tended to bend when affected by pressure differences between the cubicles. Overall, the SARS Buster's system achieved a reasonably cool environment for health care workers in the corridor. Each bed was also assigned two exhaust grilles – one at floor level and another just at bed-head level. Most bed-head level grilles did not function as expected. The challenge of the ventilation design was to remove both the small particles ($< 10\ \mu$m) and the large particles ($> 100\ \mu$m).

Dispersion of virus-laden bio-aerosols from the index patient

Although the air temperature was rather uniformly distributed in the ward, the distribution of the virus-laden bio-aerosols was not. The supply ceiling jets had ensured a rather uniform distribution of bio-aerosols in each cubicle; however, the airflows between rooms and the single virus-laden bio-aerosol source had produced a non-uniform distribution of bio-aerosol concentrations. In Figure 8, the bio-aerosol concentrations were normalized by the same reference concentration for all four simulated situations. At a height of 1.1 m, the concentration decreased as the virus-laden bio-aerosols moved away from the index patient' cubicle (Figure 8). From Figure 8, it can be seen that the concentrations at the doorstep of the patients' toilet (adjacent to the index patient's cubicle) and the store/clean room were relatively high. This meant that there was also a risk if other patients in the distant cubicles visited the toilets. The extraction fans in the store/cleaning room and in the patients' toilet seemed to have also contributed to the spread of bio-aerosols from the index patient's cubicle to the corridor and the nurses' station. This demonstrated the difficulty of controlling airflow patterns in a ward as complex as Ward 8A.

The simulated bio-aerosol distribution agreed fairly well with the limited bio-aerosol measurements performed on July 22, 2003 (Figure 8a). The measurements were performed using an bio-aerosol generator (to generate bio-aerosols with diameters between 0.1 and 10 μm) placed in one of the beds next to the index patient's bed as it was occupied. Bio-aerosol concentrations were measured in all beds in the index patient's cubicle and in the adjacent cubicle, as well as four selected beds in distant cubicles. As there was only one particle counter, bio-aerosol measurements were conducted one after another, with each point measurement taking about 2–3 min for sampling. Due to the presence of people during the measurements, some mixing may have been introduced because of their movement. The measured bio-aerosol concentrations were adjusted to exclude the background bio-aerosol level measured in each bed, and were then normalized by the concentration in the source patient's bed.

The number of beds also made a difference in the virus-laden bio-aerosol concentration, assuming that the index patient stayed in the same bed (no. 11). The overall concentration levels in the adjacent and distant cubicles were slightly lower at the time of exposure (Figure 8b) than at the time of measurement (Figure 8a). There were two possible explanations. The index patient's bed at the time of measurement (assumed) was closer to the corridor than at the time of exposure, which might have caused an easy spread of pollutants from the index patient. At the time of measurement, the index patient's bed was also closer to the supply diffuser, which made the interaction between the plume above the index patient and the supply jet stronger than that at the time of exposure.

We simulated a hypothetical situation where the flow balancing in the ward was perfect (Figure 8c). The supply airflow rate was the same for all supply diffusers at 0.31 l/s. The supply air temperature was also the same for all diffusers at 14.3°C, an average of the measured supply temperatures of all four diffusers. A slight improvement in bio-aerosol concentration was observed in both the adjacent and distant cubicles (Figure 8c). At the time of measurement, the concentrations in the adjacent cubicle were between 0.008 and 0.015, while after the flow balancing was considered, the concentration levels in the adjacent cubicle were between 0.005 and 0.015. The concentration in the distant cubicles was between 0.0015 and 0.008 at the time of measurement, and were mainly between 0.0015 and 0.005 after the flow was balanced.

The new hypothetical ventilation system was found to perform relatively well in Ward 8A (Figure 8d). The concentration in the adjacent cubicle was reduced to between 0.003 and 0.005, while in the distant cubicles to between 0 and 0.003. This presented a reasonable improvement as compared with that at the time of measurement. The bio-aerosols originating from the index patient were mostly contained in the index patient's cubicle.

Association between bio-aerosol dispersion and infection patterns

As shown in Figure 8b, bio-aerosol concentration was the highest in the index patient's cubicle at the time of exposure, followed by the adjacent cubicle, and the two distant cubicles. This bio-aerosol dispersion pattern seemed to be associated with the spatial infection pattern in Ward 8A. The number of inpatients who developed SARS in each bed is also shown in Figure 8b. The number of inpatients shown in Figure 8b added up to more than those shown in Figure 1, as some patients had occupied several beds on different days. The attack rate was the highest in the index patient's cubicle, followed closely by the adjacent cubicle. The attack rates in the two distant cubicles were low. The distant cubicle which housed Beds 25–32 had a slightly higher average concentration than its neighboring cubicle housing Beds 1–8. Correspondingly, the attack rate in the former cubicle was also slightly higher than the latter. The predicted bio-aerosol dispersion pattern at a height of 1.1 m at the time of exposure (Figure 8b) also agreed well with the spatial infection pattern of the medical students (Figure 1).

The association between the predicted bio-aerosol concentration and the spatial infection pattern suggested a probable airborne transmission route in the Ward 8A outbreak, in addition to the commonly accepted large droplet and close personal contact transmission. This finding revealed that the air distribution in Ward

8A played an important role in the SARS infection during the outbreak in March 2003 in which nearly 140 people were infected. The design of the air-conditioning system in the ward should be improved to minimize cross-infection of airborne respiratory infectious diseases such as SARS and influenza. The finding on unbalanced flow rates in supply diffusers during our on-site measurements was also important, suggesting that the testing and commissioning of an air-conditioning system for a hospital ward should be done carefully. Regular checks of the flow balancing was also necessary.

There was a general misconception among the public, i.e. that if a disease was airborne, the disease virus could be everywhere in the air of an enclosed space. With this view, any bio-aerosols generated from the evaporation of droplets produced by the index patient during coughing or sneezing, could eventually be distributed evenly in an enclosed space. From the pollutant dispersion theory, this was a very unlikely scenario, as the bio-aerosols were normally generated in one part of the room and the concentration at a distance could not be greater than that at the source at steady state. The concentration normally decayed as we moved away from the source. The concentration profile found in Ward 8A supported this argument well. If the risk of infection was a function of the virus concentration in the space, as well as (probably) the exposure time, then the risk of infection depended also on the separation distance between the virus source and the new host.

Conclusions

This paper presents a detailed air distribution study of a hospital ward during a major nosocomial outbreak in Hong Kong in March 2003. Retrospective on-site inspections and measurements of the ventilation design and air distribution system showed that the flow rates in the supply diffusers and exhaust grilles were not balanced. It was suggested that flow balancing be periodically carried out in hospital wards – say once a year. CFD simulations showed that there was an association between the concentration decay from the index patient's bed and the spatial SARS infection pattern. This provided environmental evidence of an airborne transmission route for SARS in Ward 8A.

The highly possible airborne transmission of the SARS virus in Ward 8A revealed the need for the development of improved ventilation and air-conditioning systems in a SARS isolation ward. Such a system design should effectively reduce the risk of cross-infection between patients and between a patient and health care workers. The outbreak in Ward 8A, which was in a general hospital and could house more nearly 40 patients, demonstrated the cross-infection risks of respiratory infectious diseases in hospitals if a potential highly infectious patient was not identified and isolated.

One question often asked by ventilation engineers was why effective displacement ventilation could not be used in general hospital wards. This question has already been addressed by Friberg et al. (1996). They found that displacement ventilation performed better at removing small particles (< 0.3 μm) than conventional mixing systems, but the displacement system also yielded two- to threefold higher air and surface bacterial counts, and it could not efficiently remove the larger bacteria-carrying particles.

Our example simulation, which extended the SARS Busters' design for an isolation room for Ward 8A, demonstrated that there was room for improvement to minimize cross-infection in large general hospital wards. The SARS-Busters' design for multi-bed isolation wards was not recommended for general medical wards, although, as shown here, the ventilation performance in Ward 8A could have been improved with this design. The SARS Busters' system was too complex when directly applied to a general ward with multiple cubicles, and there were also significant space and financial cost implications. Further investigation should be carried out to simplify and optimize the SARS Busters' design for use in large general hospital wards, and to develop alternative effective systems.

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