2015

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Published in *The Annals of Occupational Hygiene* 59:9 (2015), pp. 1190-1199. doi 10.1093/annhyg/mev048

Submitted 30 March 2015; revised 15 June 2015; accepted 17 June 2015.

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Abstract

Most studies on the transmission of infectious airborne disease have focused on patient room air changes per hour (ACH) and how ACH provides pathogen dilution and removal. The logical but mostly unproven premise is that greater air change rates reduce the concentration of infectious particles and thus, the probability of airborne disease transmission. Recently, a growing body of research suggests pathways between pathogenic source (patient) and control (exhaust) may be the dominant environmental factor. While increases in airborne disease transmission have been associated with ventilation rates below 2 ACH, comparatively less data are available to quantify the benefits of higher air change rates in clinical spaces. As a result, a series of tests were conducted in an actual hospital to observe the containment and removal of respirable aerosols (0.5–10 μm) with respect to ventilation rate and directional airflow in a general patient room, and, an airborne infectious isolation room. Higher ventilation rates were not found to be proportionately effective in reducing aerosol concentrations. Specifically, increasing mechanical ventilation from 2.5 to 5.5 ACH reduced aerosol concentrations only 30% on average. However, particle concentrations were more than 40% higher in pathways between the source and exhaust as was the suspension and migration of larger particles (3–10 μm) throughout the patient room(s). Computational analyses were used to validate the experimental results, and, to further quantify the effect of ventilation rate on exhaust and deposition removal in patient rooms as well as other particle transport phenomena.

Keywords: bioaerosols; CFD; hospital, ventilation

Introduction

In addition to occupant comfort, heating, ventilation, and air conditioning (HVAC) systems provide continuous indoor air quality (IAQ). As a result, hospital HVAC is generally not load-driven, but is predicated on providing adequate ventilation air to maintain a wide range of directional airflow relationships (from cleaner to less clean spaces) and air change rates to contain, dilute and remove hazards such a volatile medical gases, particulates, and airborne disease transmission (Grosskopf and Mousavi, 2014). Airborne disease refers to any disease that is caused by pathogens such as viruses, bacteria, and fungi, and, is transmitted through the air. Airborne disease transmission occurs when pathogenic microorganisms be-

come aerosolized on small particles or droplets (≤10 μm) and spread from the environment or host individual to other susceptible individuals, usually through respiratory activity. Infection may occur when pathogenic organisms capable of producing disease enter a vulnerable host site in sufficient numbers to survive and multiply.

Although one-third of healthcare-acquired infections may involve airborne transmission at some point (Kowalski, 2007), only a few diseases currently require infectious airborne isolation. To reduce both the concentration and time patients and healthcare workers are exposed to pathogenic microorganisms, ASHRAE Standard 170 and several other guidelines recommend 6–12 ACH for infectious isolations rooms (AIA, 2006; Siegel *et al.*, 2007; ASHRAE, 2008; Atkinson *et al.*, 2009). Although higher air change rates can better dilute contaminant concentrations within a patient room, air changes alone have not proven to reduce the risk of airborne cross infection (Marshall *et al.*, 1996; Novoselac and Srebric, 2003; Walker *et al.*, 2007; Johnson *et al.*, 2009; Memarzadeh and Xu, 2011). For this, new research has begun to look beyond air change rates to examine the effects that other factors such as supply and exhaust location, door position and motion, spatial orientation, surface composition, temperature, humidity, and air distribution patterns have on particle migration in clinical spaces.

Just as protection between clinical spaces depends on directional airflow, protection within clinical spaces also depends on directional airflow. The results of several recent studies suggest that the most important factor contributing to contaminant transmission in enclosed mechanically ventilated spaces is the path between the contaminant source and exhaust. Ideally, airflows in patient rooms should be directional and laminar (Hyttinen *et al.*, 2011) between supply, source, and exhaust. When the exhaust is located away from the contaminant source, is influenced by nearby supply air, or, the source is outside of the directional airflow between supply and exhaust, contaminants migrate to other places in the patient room (Memarzadeh and Xu, 2011). In cases where downward ventilation design achieves laminar, directional airflow, the physical and thermal effects of patients, healthcare workers, and equipment can cause unintended mixing (Qian *et al.*, 2006).

Airflow velocities necessary to achieve high air change rates invariably produce turbulent airflows. Turbulent airflows associated with high air change rates may not only interfere with directional airflow within clinical spaces, but may also breakdown containment between clinical spaces (Rydock and Eian, 2004). To validate the findings of these and other similar studies, a series of experimental tests were conducted in an actual hospital to observe the effectiveness of air change rates and supply and exhaust location to contain, dilute, and remove respiratory aerosols in general and within isolation patient rooms designed for mixing ventilation. Aerosol containment between patient rooms and corridors was also observed relative to air change rate and directional airflow, door motion and position, spatial orientation, and air distribution patterns. Numerical analyses were used to validate the empirical results, and to further quantify other particle transport phenomena in general and isolation patient rooms.

Methods

Experimental method

A total of four experimental tests were conducted; two each in a general patient room and an infectious isolation room. In the general patient test room, flow hood measurements verified that ventilation air (40.1 l s–1) and exhaust air (40.6 l s–1) were nearly balanced, producing 2.5 mechanical air changes per hour (ACH), and, a neutral air pressure relationship with respect to the corridor. In the isolation patient test room (Fig. 1), flow hood measurements verified that exhaust air (102.9 l s–1) exceeded ventilation air (64.7 l s–1), producing 5.5 ACH, and, a negative 2.5 Pa air pressure relationship with respect to the corridor. The spatial uniformity of ventilation was verified by means of ASTM E741 (American Society for Testing and Materials International, 2008) tracer gas (SF6) dilution in all patient rooms and adjacent corridors.

To simulate a respiratory aerosol, a synthetic oil (poly aliphatic olefin) ~84.7% of the density of water was continuously aerosolized at a rate of 15 mg 0.4 L−1 of air per second using a NUCON SN-10 pneumatic aerosol generator. The aerosolization rate was roughly twice the respiratory rate of a healthy human at rest (0.7 l per breath, 16–18 breaths per minute) and consistent with other studies using synthetic

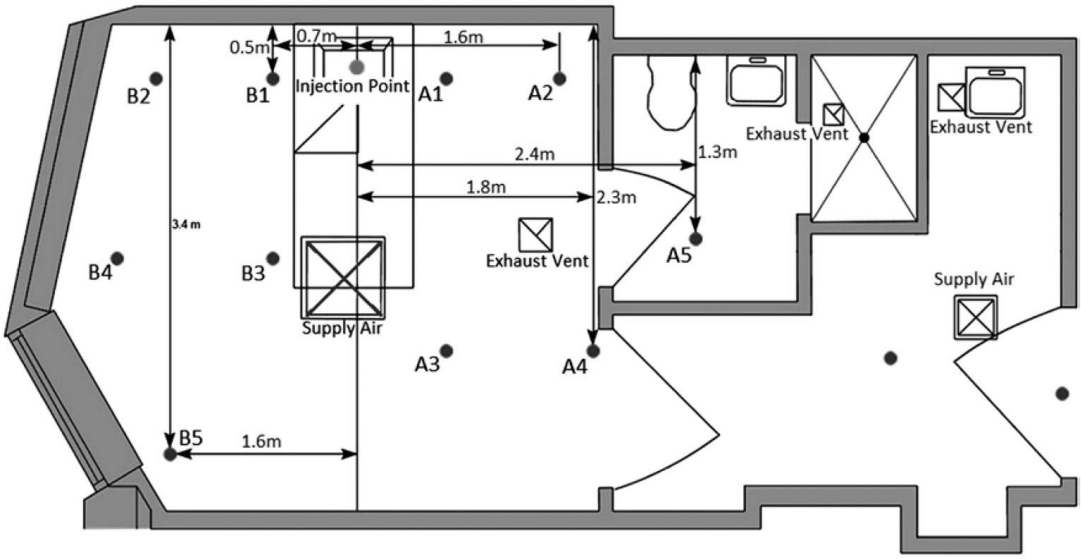

Figure 1 Aerosol sampling locations in the isolation patient room.

respiratory aerosols (Wan *et al.*, 2007; Chao *et al.*, 2008). The aerosol, with mean aerodynamic diameters (*d*a) of 0.5–10 μm, was released at an approximate height of a patient lying at rest (0.8 m). The particle size distribution used for this study represents the size range of desiccated respiratory droplets or airborne 'droplet nuclei' (Tang *et al.*, 2006) and was again consistent with other studies (Papineni and Rosenthal, 1997; Fennelly *et al.*, 2004; Nicas *et al.*, 2005; Xie *et al.*, 2007; Yang *et al.*, 2007) which suggest that human respiratory activity (e.g. coughing, sneezing, etc.) generates as many as 40 000 droplets of 0.5–12 μm diameter (Cole and Cook, 1998). With settling velocities <1 m h-1 in still air, particles <5 μm can remain airborne almost indefinitely (Qian *et al.*, 2006) and are most capable of producing infection in the deep, alveolar region of the lung. Droplets with a mass median aerodynamic diameter of 10 μm are widely considered as the upper size limit for airborne transmission (Hyttinen *et al.*, 2011).

Particle size measurements (particles l^{-1}) were collected using a NUCON F-1000-DD light scattering photometric aerosol detector at a total of 10 sampling locations in each test room (Fig. 1). The aerosol detector was a six-channel instrument with ±1% reading accuracy and 0.0001 μg l^{-1} threshold sensitivity. Each sampling location consisted of three sampling points at 0.6, 1.2, and 1.8 m above the patient room floor (Fig. 2). Air samples from each sampling point were drawn at 30 s intervals for a total of 30–40 min each. In addition, two Lighthouse HH-3016-IAQ portable particle size counters were positioned in the center of the general patient bathroom (location of room exhaust) at a sampling height of 1.2 m, and, in the corridor outside the patient room above the entry door. Two additional portable particle size counters were positioned in the center of the isolation patient anteroom at a sampling height of 1.2 m, and, in the corridor outside the anteroom room above the entry door. All equipment and instrumentation was calibrated prior to testing according to ASME AG-1 and ASHRAE 52.2.

At the start of testing in the general patient test room, the entry door was closed and the bathroom door was open. At the start of testing in the isolation patient test room, the doors to the anteroom and the isolation room were closed and the bathroom door was open. In both test rooms, concentrations of ambient airborne particles were sampled for 30 min prior to aerosol injection. After ~3 h of sampling in the general patient test room, the entry door was opened for the remainder of testing. Thirty minutes later, the bathroom door was closed for the remainder of testing. After ~3.5 h of sampling in the isolation patient test room, the door from the anteroom to the isolation room was opened for the remainder of testing. Thirty minutes later, the door leading to the anteroom from the corridor was opened for the remainder of testing. For both general patient and isolation room tests, aerosol injection was terminated 30 min after the sec-

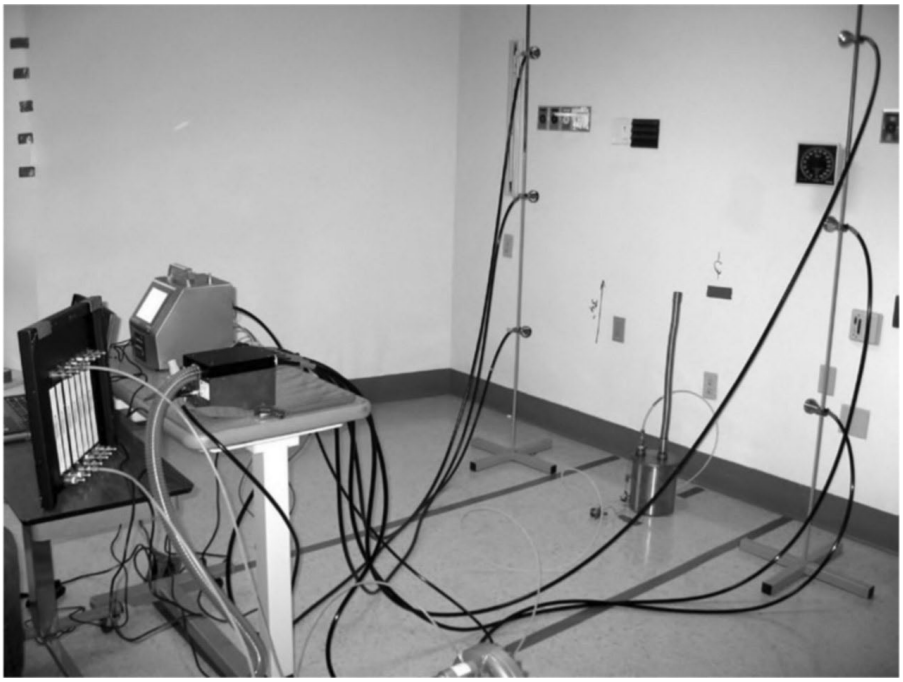

Figure 2. Aerosol generator and particle sampling equipment used in the general and isolation patient test rooms. Sampling locations A1 and B1 shown at 0.6, 1.2, and 1.8 m sampling heights, respectively. (Grosskopf and Mousavi, 2014)

ond door position change and samples collected for an additional 30 min to determine the time necessary to ventilate the test rooms to background levels. The intent of this test procedure was to observe the effectiveness of directional airflow and ventilation rates to contain, dilute, and remove respiratory aerosols, and, evaluate the effects of door position and personnel movement on particle transport phenomena in patient room environments.

Computational method

Computational fluid dynamic (CFD) analyses were used to validate experimental results, and, to further explore the particle transport phenomena in patient room environments. Using ANSYS Fluent software, a computational model was constructed to replicate the experimental airflow pattern inside the isolation room. The isolation room geometry (Fig. 1) was used and the boundary conditions were similar to those recorded during the tests (e.g. the inlet/outlet flow rates). Assuming airflows are generally turbulent (Yakhot *et al.*, 1992; Novoselac and Srebric, 2002; Chen and Zhao, 2010), the realizable K-Ɛ model was used to model the turbulence. The tiny-box method (Srebric and Chen, 2011) was used to model the supply diffuser by introducing a 3% initial turbulence (Azad, 1996).

Furthermore, air was deemed to be a perfect gas, therefore its density changed with the calculated pressure and temperature. Moreover, the 'make-up' air needed to balance the difference between inlet and outlet flow rates was assumed to enter through the space underneath the isolation room door. Other infiltration mechanisms such as infiltration through wall cracks and windows were considered negligible. The SIMPLE algorithm (Patankar and Spalding, 1972) was employed to solve the Navier–Stokes equations coupled with the energy equation through an iterative process. Upon obtaining the flow pattern, an Eulerian–Lagrangian approach was utilized to analyze the particle motion within the room. In this approach, particles are assumed to be solid, nondeformable entities whose motion is determined by the forces exerted on them. The Brownian force and the Saffman's lift force were exerted on particles while other forces such as the pressure gradient and Basset force were neglected (Zhao *et al.*, 2004). Particles were presumed to 'trap' when colliding with solid surfaces (deposition), and escape from the exhaust fans (removal). A Discrete Random Walk (DRW) model was also used to factor in the effect of fluctuating components of the velocity due to the existing turbulence (Hathway *et al.*, 2011).

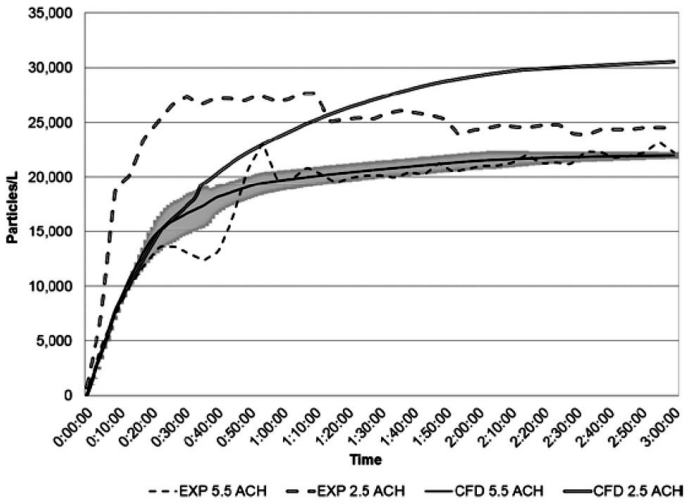

Figure 3. Average test room particle concentration relative to air change rate per hour (1.0 μm), experimental (EXP) versus computational (CFD).

In order to validate the CFD results by the experimental outcomes, both clusters of data were put in an equivalent form. Also, to merely analyze the timedependent trend of particle concentration, the spatial variable was integrated out, and consequently, particle concentration of the entire room was depicted by time (Fig. 3). Similarly, the average concentrations of six concurrent sample locations embodied the integration process for the test segment. Experimental data suggested that the average particle concentration increased by the onset of injection until it reached the steady state condition. Turbulence of the flow and the Brownian motion of particles brought about a narrow distribution of the numerical results (shaded area encompassing the CFD results) (Fig. 3). Indeed, the CFD model was executed five times to manifest the effect of stochastic elements on the particle distribution.

To quantify the similarity between the two sets of data, a paired two sample T-test was used. Since the data sets were non-permutable, they had to be compared at each corresponding time. The T-test result suggests that there is no statistically meaningful difference between the two data at a 99% confidence level ($P < 0.001$). The Pearson correlation coefficient was also calculated (95.7%). Therefore, there was no statistically significant difference between the experimental and CFD results.

Results

In the general patient test room, concentrations of particles >1.0 μm decreased 6.1%, on average, every ~0.5 m from the aerosol injection point ($r2 = -0.63$; Table 1). Concentrations of particles >1.0 μm at 'A' series sampling locations (i.e. pathway between supply and exhaust) were 23.9% greater, on average, than corresponding concentrations of particles at 'B' series sampling locations at the same sampling height. Furthermore, concentrations of particles >1.0 μm were found to be greater at 'A' series 1.8 m sampling heights when compared with 0.6 m sampling heights, suggesting that particles >1.0 μm may remain airborne longer within the airflow pathway between the supply and exhaust air. Outside of the pathway, however, concentrations of particles >1.0 μm were found to be greater at lower sampling heights, suggesting the presence of gravitational settling. In contrast, concentrations of particles <1.0 μm remained relatively constant, regardless of time and distance from the aerosol injection point, throughout testing until the entry door to the corridor was opened. Particles <1.0 μm appeared to disperse randomly and uniformly within the general patient test room under the influences of mechanical airflow, kinetic particle movement (e.g. 'Brownian motion'), or both.

Next, concentrations of particles in the general patient test room were observed with respect to door position and motion. The maximum temperature difference was observed to be 1oC between two adjacent spaces (Table 2). Although this is not a large temperature gradient, it can cause two-way air exchange between rooms due to a relatively large opening area (Chen *et al.*, 2011). This phenomenon, in addition to the turbulence created by the door opening motion

Table 1. Average change in particle concentration relative to particle size, sample height, and sample location in test rooms.

Test room	Height (m)	Particles <1.0 μm		Particles >1.0 μm	
		'A' series (%)	'B' series (%)	'A' series (%)	'B' series (%)
General patient room	0.6	−1.2	−0.4	−4.7	−4.0
	1.2	−0.6	−0.1	−7.6	−5.4
	1.8	0.0	0.4	−9.5	−5.3
Isolation room	0.6	5.5	3.2	11.4	6.3
	1.2	4.8	3.7	4.1	7.9
	1.8	4.4	5.6	9.0	10.6

Table 2. Air Temperature across the doors.

	Test 1, temperature (ºC)/SD	Test 2, temperature (ºC)/SD
Isolation room	21.8°C/0.3	23.3°C C/ 0.4
Anteroom	20.8°C /0.4	22.8°C C/ 0.2
Corridor	21.2°C /0.2	22.0°C C/ 0.2
Patient room	20.3°C /0.4	23.0°C C/ 0.9
Corridor	20.8°C /0.4	22.2°C C/ 0.2

appeared to allow a small, intermittent release of particles into the corridor (See Supplementary Fig. S1 at *Annals of Occupational Hygiene* online [requires subscription or purchase]).

When the entry door was left open (t = 3:00 h), however, a significant and sustained release of particles from the test room to the corridor was observed despite the neutral air pressure relationship between the general patient room and corridor. Approximately 30 min after the entry, door to the corridor was left open, the general patient bathroom door was closed, causing concentrations of particles in the corridor to increase again. These data suggest that when closed, the bathroom door impinged exhaust air ventilation (located in the bathroom), causing the general patient test room to pressurize and release aerosol into the corridor.

Within the isolation patient test room, concentrations of particles >1.0 μm increased 8.2%, on average, every ~0.5 m from the aerosol injection point ($r2$ = 0.71; Table 1). Concentrations of >1.0 μm particles at 'A' series sampling locations were 40.6% greater, on average, than corresponding concentrations of particles at 'B' series sampling locations at the same sampling height. Concentrations of particles >1.0 μm were found to be greater at both 'A' and 'B' series 1.8 m sampling heights when compared with 0.6 m sampling heights. To a lesser degree, concentrations of particles <1.0 μm also increased, 4.5% on average, every ~0.5 m from the aerosol injection point ($r2$ = 0.75). Concentrations of <1.0 μm particles at 'A' series sampling locations were 9.3% greater, on average, than corresponding concentrations of particles at 'B' series sampling locations at the same sampling height.

Concentrations of particles were also observed with respect to door position and motion in the isolation patient test room. The turbulence created by the door opening motion appeared to allow small, intermittent

release particles into the anteroom (See Supplementary Fig. S2 is available at *Annals of Occupational Hygiene* online) but not the corridor. When the inner anteroom door was left open (t = 3:30 h), however, a significant and sustained release of particles from the test room to the anteroom was observed despite the neutral air pressure relationship between the anteroom and isolation room. Correspondingly, concentrations of particles increased in the corridor although only briefly. Approximately 30 min after the inner anteroom door to the isolation room was left open, the outer anteroom door to the corridor was also left open. Again, concentrations of particles increased only briefly in the corridor, suggesting that the negative air pressure relationship between the anteroom and corridor (e.g. inward airflow from corridor to anteroom) was effective in containing the release of aerosol from the isolation patient test room into the corridor.

Finally, concentrations of particles were observed with respect to ventilation air change rate in both general patient and isolation patient test rooms. Specifically, 2.5 ACH were observed in the general patient room during testing compared to 5.5 ACH observed in the isolation patient room. By comparing concentrations of particles in general patient and isolation test rooms, air change rates were not found to be proportionately effective in reducing aerosol concentrations. Increasing ventilation rates from 2.5 to 5.5 ACH reduced aerosol concentrations only 30% on average (Fig. 3), or, 22.6% and 38.5% for <1.0 and >1.0 μm particles, respectively (Table 3).

This finding, however, ignores the spatial and temporal differences in each room and assumes a steady-state, well-mixed condition in both rooms where ventilation rate, particle generation rate, and particle concentration in the supply are the same. Therefore, CFD models were developed and validated to appraise the particles fate and transport within the isolation room relative to ventilation rate. Each particle was followed until reaching its destiny: removal by the exhaust fans, or deposition onto the room surfaces. Most particles were found to have a distinct destiny; however, few particles (e.g. incomplete) were entrained in a flow vortex and remained suspended almost indefinitely. The results revealed that the removal rate increased disproportionately relative to ventilation rate (Table 4). Conversely, deposition onto the floor increased greatly under 2.5 ACH suggesting the dominance of gravitational settling under lower ventilation rates.

Particles tend to settle out more effectively under lower ventilation rate indicating that the upward air movement enhanced suspension of particles. The height distribution of particles further substantiated a propensity to ascend under higher ventilation rate (Fig. 4). Particles average height was 1.69 m (σ = 0.82) for 5.5 ACH compared to 1.32 m (σ = 0.96) for 2.5 ACH. Also, the maximum residence time and the distance traveled by particles decreased 30 and 17% (Table 5), respectively, when the ventilation rates roughly doubled.

Table 3. Average particle concentration [particles l−1] relative to particle size and air change rate per hour in general and isolation patient test rooms.

Ventilation	0.5 μm	0.7 μm	1.0 μm	3.0 μm	5.0 μm
2.5 ACH	10,160	5,179	12,242	270	7
5.5 ACH	8917	3469	8056	143	4
Change	−12%	−33%	−34%	−47%	−34%

Table 4. Particle deposition and exhaust air removal rates in general (2.5 ACH) and isolation (5.5 ACH) patient test rooms.

Ventilation rate	Removal (exhaust)		Deposition			Incomplete(%)	Total (%)
	Isolation room (%)	Bathroom (%)	Floor (%)	Ceiling (%)	Wall (%)		
5.5 ACH	43.4	8.7	17.6	8.2	18.7	3.4	100
2.5 ACH	26.5	8.1	26.6	11.2	22.5	5.0	100

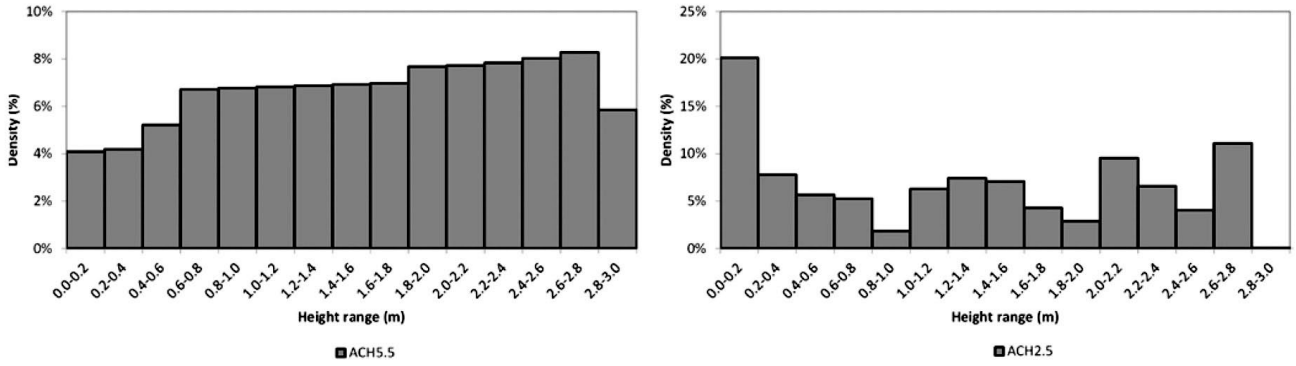

Figure 4. Particle height distribution relative to ventilation rate.

Table 5. Average residence time and distance travelled by particles.

Ventilation rate	Average height (m), SD	Average residence time (min), SD	Average distance traveled (m), SD
5.5 ACH	1.69 (0.8)	17.54 (33.5)	106.64 (87.2)
2.5 ACH	1.32 (0.9)	25.26 (56.5)	128.85 (94.8)
Change	−28.1%	30.5%	17.2%

Discussion

Particles <1.0 μm were found to exhibit different aerodynamic behaviors when compared to particles >1.0 μm, as did aerosols subject to different environmental conditions within a general patient test room and isolation patient test room. Concentrations of particles >1.0 μm decreased with respect to distance from the aerosol injection point (e.g. 'patient') in the general patient test room. In contrast, concentrations of particles >1.0 μm increased with respect to distance in the isolation patient test room. Within the general patient test room, the tendency for concentrations of larger particles to decay with respect to time and distance may be explained by lower airflow rates and the volumetric dominance of downward supply air ventilation, thus enabling gravitational settling and surface deposition. Conversely, the tendency for particles to remain suspended within the isolation patient test room may be explained by higher airflow rates and the volumetric dominance of upward exhaust air ventilation (Table 4). Furthermore, higher concentrations of particles were observed at higher sampling heights in the isolation patient room, especially particles >1.0 μm. Accordingly, the CFD results suggested that the average height of particles increased within the isolation patient room. Although increasing the airflow rate resulted in a decrease in residence time and distance traveled by particles, this change was not commensurate with the extra energy required for higher flowrates (Table 5). Similarly, particle concentrations did not proportionally decrease by introducing higher air change rates (Fig. 3). Although a study of 1,289 healthcare workers in 17 Canadian hospitals found the risk of tuberculosis transmission 3.4 times higher in patients rooms with <2.0 ACH when compared to patient >2.0 ACH (Menzies, 2000), empirical and numerical test results suggest that turbulence created by higher air change rates could reduce the benefits of bioaerosol removal by suspending infectious particles within breathing zone (1.2–1.8 m).

Air pressure relationships, door position, and door motion were also found to have a significant effect on aerosol behavior in both patient room tests. A neutral air pressure relationship between the general patient room and corridor, and, the isolation patient room and anteroom, was found to be effective in containing both <1.0 and >1.0 μm particles when the door separating these spaces was closed. Door motion, however, was found to cause a transient breakdown in aerosol containment, allowing the intermittent release of both <1.0 and >1.0 μm particles from the general patient

room to the corridor, and, from the isolation patient room to the anteroom. An analysis of the door-opening motion indicates that even a negative pressure relationship can be temporarily reversed if the door-opening motion is too rapid. The exchange volume of air produced by the door-opening motion is comparable to the swept volume of the door (~3 m^3). A person with a forward projected area of 0.8 m^2 entering the patient room at 1m/s can further generate a 'body wake' of approximately 4 m^3 (Tang *et al.*, 2005; Eames *et al.*, 2009; Tang *et al.*, 2013). Together, as much as 5% of the isolation room volume can be transported to the corridor by a healthcare worker entering or exiting a patient room despite a −2.5 Pa pressure difference (Eames *et al.*, 2009).

When left open, a significant and sustained release of both <1.0 and >1.0 μm particles was observed across the neutral airflow boundary separating the general patient room from the corridor, and, the isolation patient room and anteroom. In contrast, a negative air pressure relationship between the anteroom and corridor was found to be effective in containing >1.0 μm particles regardless of door position. Particles <1.0 μm, however, were found capable of escaping into the corridor when the air pressure of isolation room became positive with respect to the anteroom, despite inward airflow from corridor to anteroom and closed doors in both anteroom and isolation room. Further analyses suggest that if a temperature difference exists between the isolation room and corridor, convection may force warmer air from the isolation room out into the corridor and to nearby patient rooms even if the entry door is closed (Tang *et al.*, 2005; Chen *et al.*, 2011). A cluster sample of 346 patients with acquired immunodeficiency syndrome (AIDS) found that 21 nosocomial tuberculosis infections occurred in a total of 16 patient rooms that were located two rooms or less away from index cases. In four of these rooms, inward airflow from the corridor to the patient room was observed at the bottom of the doorway while outward airflow from the patient room to the corridor was observed at the top of the doorway (Edlin *et al.*, 1992). Moreover, using numerical modeling Memarzadeh et al. (Memarzadeh and Xu, 2011) showed that the contaminant dilution via ventilation is not proportionate to the ventilation rate which is consistent with the present findings of this work.

The results of this study suggest that negative pressurization recommended by healthcare ventilation standards such as ASHRAE 170-2008 (ASHRAE, 2008) are effective in containing aerosol transport. However, results also suggest that higher air change rates may not be proportionately effective in removing infectious aerosols from patient rooms, and, may have the unintended consequence of increasing breathing zone exposure to particles suspended in turbulent airflow.

Supplementary data is held at http://annhyg.oxfordjournals.org/ (requires subscription or purchase).

Funding — This research was partially funded by the U.S. Department of Veterans Affairs (VA) Veterans Health Administration in response to the 2009 Swine Flu Pandemic. The VA has released this data to be publically disseminated under the Freedom of Information Act (FOIA ref. 11-01558-F).

Declaration — As the authors of this article, we attest to its originality and accuracy and do hereby certify that the authors have no known conflict of interest and are in full compliance with the submission declaration.

References

AIA. (2006) *Guidelines for Design and Construction of Health Care Facilities*. Second edition, Washington, DC: The American Institute of Architects. American Society for Testing and Materials International. (2008) *ASTM E 741 Standard Test Method for Determining Air Change in a Single Zone by Means of a. ASTM International Standard*, pp. 1–17.

ASHRAE. (2008) *Standard, 170–2008 Ventilation of Health Care Facilities*. Atlanta, GA: ASHRAE.

Atkinson J, Chartier Y, Pessoa-Silva CL *et al.* (2009) *Natural ventilation for infection control in health-care settings*, World Health Organization. ISBN 978 92 4 154785 7

Azad RS. (1996) Turbulent flow in a conical diffuser: A review. *Exp Thermal Fluid Sci*; 13: 318–37.

Chao CYH, Wan MP, Sze To GN. (2008) Transport and removal of expiratory droplets in hospital ward environment. *Aerosol Sci Technol*; 42: 377–94.

Chen C, Zhao B, Yang X *et al.* (2011) Role of twoway airflow owing to temperature difference in severe acute respiratory syndrome transmission: revisiting the largest nosocomial severe acute respiratory syndrome outbreak in Hong Kong. *J R Soc Interface*; 8: 699–710.

Chen C, Zhao B. (2010) Some questions on dispersion of human exhaled droplets in ventilation room: answers from numerical investigation. *Indoor Air*; 20: 95–111.

Cole EC, Cook CE. (1998) Characterization of infectious aerosols in health care facilities: An aid to effective engineering controls and preventive strategies. *Am J Infect Control*; 26: 453–64.

Eames I, Shoaib D, Klettner CA *et al.* (2009) Movement of airborne contaminants in a hospital isolation room. *J R Soc Interface*; 6(Suppl. 6): S757–66.

Edlin BR, Tokars JI, Grieco MH *et al.* (1992) An outbreak of multidrug-resistant tuberculosis among hospitalized patients with the acquired immunodeficiency syndrome. *N Engl J Med*; 326: 1514–21.

Fennelly KP, Martyny JW, Fulton KE *et al.* (2004) Coughgenerated aerosols of Mycobacterium tuberculosis: a new method to study infectiousness. *Am J Respir Crit Care Med*;,169: 604–9.

Grosskopf KR, Mousavi ES. (2014) Ventilation and transport bioaerosols in health-care environments. *ASHRAE J*; 56: 22–31.

Hathway E, Noakes CJ, Sleigh PA *et al.* (2011) CFD simulationof airborne pathogen transport due to human activities. *Build Environ*; 46: 2500–11.

Hyttinen M, Rautio A, Pasanen P *et al.* (2011) Airborne infection isolation rooms - A review of experimental studies.*Indoor Built Environ*; 20: 584–94.

Johnson DL, Lynch RA, Mead KR. (2009) Containment effectiveness of expedient patient isolation units. *Am J Infect Control*; 37: 94–100.

Kowalski WJ. (2007) Air-treatment systems for controlling hospital-acquired infections. *HPAC Eng*; 79: 28–48.

Marshall JW, Vincent JH, Kuehn TH *et al.* (1996) Studies of ventilation efficiency in a protective isolation room by the use of a scale model. *Infect Control*; 17: 5–10.

Memarzadeh F, Xu W. (2011) Role of air changes per hour (ACH) in possible transmission of airborne infections. *Build Simul*; 5: 15–28.

Menzies D. (2000) Hospital ventilation and risk for tuberculous infection in canadian health care workers. *Ann Intern Med*; 133: 779.

Nicas M, Nazaroff WW, Hubbard A. (2005) Toward understanding the risk of secondary airborne infection: emission of respirable pathogens. *J Occup Environ Hyg*; 2: 143–54.

Novoselac A, Srebric J. (2002) A critical review on the performance and design of combined cooled ceiling and displacement ventilation systems. *Energy Build*; 34: 497–509.

Novoselac A, Srebric J. (2003) Comparison of air exchange efficiency and contaminant removal effectiveness as IAQ indices. *ASHRAE Trans*; 109: 339–49.

Papineni RS, Rosenthal FS. (1997) The size distribution of droplets in the exhaled breath of healthy human subjects. *J Aerosol Med*; 10: 105–16.

Patankar S, Spalding D. (1972) A calculation procedure for heat, mass and momentum transfer in three-dimensional parabolic flows. *Int J Heat Mass Transfer*; 15: 1787–1806.

Qian H, Li Y, Nielsen PV *et al.* (2006) Dispersion of exhaled droplet nuclei in a two-bed hospital ward with three different ventilation systems. *Indoor air*; 16: 111–28.

Rydock JP, Eian PK. (2004) Containment testing of isolation rooms. *J Hosp Infect*; 57: 228–32.

Siegel JD, Rhinehart E, Jackson M *et al.* (2007) 2007 guideline for isolation precautions : preventing transmission of infectious agents in healthcare settings *Am J Infect Control*; 35: S65–S124.

Srebric J, Chen Q. (2011) Simplified numerical models for complex air supply diffusers. *HVAC&R Res*; 8: 227–294.

Tang JW, Nicolle A, Pantelic J *et al.* (2013) Different types of door-opening motions as contributing factors to containment failures in hospital isolation rooms. *PloS One*; 8: e66663.

Tang JW, Eames I, Li Y *et al.* (2005) Door-opening motion can potentially lead to a transient breakdown in negative-pressure isolation conditions: the importance of vorticity and buoyancy airflows. *J Hosp Infect*; 61: 283–6.

Tang JW, Li Y, Chan PK *et al.* 2006. Factors involved in the aerosol transmission of infection and control of ventilation in healthcare premises. *J Hosp Infect*; 64: 100–14.

Walker JT, Hoffman P, Bennett AM *et al.* (2007). Hospital and community acquired infection and the built environment –design and testing of infection control rooms. *J Hosp Infect*; 65: 43–9.

Wan MP, Chao CYH, Ng YD *et al.* (2007) Dispersion of expiratory droplets in a general hospital ward with ceiling mixing type mechanical ventilation system. *Aerosol Sci Technol*; 41: 244–58.

Xie X, Li Y, Chwang AT *et al.* (2007) How far droplets can move in indoor environments--revisiting the Wells evaporation-falling curve. *Indoor Air*; 17: 211–25.

Yakhot V, Orszag SA, Thangam S *et al.* (1992) Development of turbulence models for shear flows by a double expansion technique. *Phys Fluids A Fluid Dyn*; 4: 1510.

Yang S, Lee GW, Chen CM *et al.* (2007) The size and concentration of droplets generated by coughing in human subjects. *J Aerosol Med*; 20: 484–94.

Zhao B, Zhang Y, Li X *et al.* (2004) Comparison of indoor aerosol particle concentration and deposition in different ventilated rooms by numerical method. *Build Environ*; 39: 1–8.