The ventilation of multiple-bed hospital wards: Review and analysis

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Background: Although the merits of ventilating operating theatres and isolation rooms are well known, the clinical benefits derived from ventilating hospital wards and patient rooms are unclear. This is because relatively little research work has been done in the ventilation of these areas compared with that done in operating theatres and isolation rooms. Consequently, there is a paucity of good quality data from which to make important decisions regarding hospital infrastructure. This review evaluates the role of general ward ventilation to assess whether or not it affects the transmission of infection.
Methods: A critical review was undertaken of guidelines in the United Kingdom and United States governing the design of ventilation systems for hospital wards and other multibed rooms. In addition, an analytical computational fluid dynamics (CFD) study was performed to evaluate the effectiveness of various ventilation strategies in removing airborne pathogens from ward spaces.
Results: The CFD simulation showed the bioaerosol concentration in the study room to be substantially lower (2467 cfu/m^3) when air was supplied and extracted through the ceiling compared with other simulated ventilations strategies, which achieved bioaerosol concentrations of 12487 and 10601 cfu/m^3, respectively.
Conclusions: There is a growing body of evidence that the aerial dispersion of some nosocomial pathogens can seed widespread environmental contamination, and that this may be contributing to the spread infection in hospital wards. *Acinetobacter* spp in particular appear to conform to this model, with numerous outbreaks attributed to aerial dissemination. This suggests that the clinical role of general ward ventilation may have been underestimated and that through improved ward ventilation, it may be possible to reduce environmental contamination and thus reduce nosocomial infection rates. (Am J Infect Control 2008;36:250-9.)

Although the merits of ventilating operating theatres[1-5] and isolation rooms[3,6] are well known, the clinical benefits derived from ventilating hospital wards and patient rooms are unclear. This is because relatively little research work has been done on the ventilation of these areas compared with operating theatres and isolation rooms. Consequently, there is a paucity of good-quality data from which to make important decisions regarding hospital infrastructure. Indeed, with respect to general ward ventilation, much of what has been written has tended to focus on the interpretation of building codes and regulations rather than addressing fundamental issues regarding the clinical role of ward ventilation. In light of this situation, we conducted this review to evaluate the role of general ward ventilation and to assess whether or not it affects the transmission of infection in health care facilities. We evaluate the advantages and disadvantages of the various approaches taken and compare these with current thinking regarding the spread of infection in hospital wards.

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WARD VENTILATION

Although a plethora of guidelines on the ventilation of health care facilities have been published,[7-10] the vast majority of these are concerned with specialist facilities, such as operating theatres, isolation rooms, and bronchoscopy suites, where the risks associated with the airborne transmission of infection are well characterized. In comparison, guidelines regarding the ventilation of general ward spaces, patient rooms, and intensive care wards are much sparser and often vague in nature. For example, in the United Kingdom, National Health Technical Memorandum HTM 2025 (Design Considerations, Ventilation in Health Care Premises) makes little reference to the ventilation of clinical spaces other than operating theatres.[7] Indeed, other than encouraging the use of full fresh air systems, HTM 2025 specifies no criteria for the ventilation of ward spaces. In an era where hospital-acquired infection (HAI) is a major worldwide problem, this may seem to be a surprising omission. Ward ventilation could play an important role in controlling the spread of HAI, although there is a generally held view that most nosocomial infections are transmitted by the contact route (ie, through the hands of health care workers).[11] Indeed, only a few nosocomial diseases of a bacterial or fungal etiology, such as tuberculosis

(TB), legionnaire's disease, and pulmonary aspergillosis, are readily accepted as being transmitted by an airborne route. Consequently, ward ventilation systems are generally specified in terms of providing patient comfort and minimizing energy costs, rather than for clinical reasons. In short, ward ventilation is perceived as having little impact on the transmission of HAI and thus is not rigorously specified. Notwithstanding this, there is growing evidence[12-14] indicating that airborne pathogens may play a greater role in the spread of infection within wards than hitherto expected. If this is the case, then the potential of ward ventilation systems to control infection may have been greatly underestimated, and there is a need to reevaluate the basis on which such systems are specified.

SINGLE- AND MULTIPLE-BED ROOMS

It is impossible to address the issue of ward ventilation without first considering the nature of patient rooms. In many parts of the world, including the United Kingdom, it is common practice to have multiple-bed wards, often subdivided into bays containing 4 or 6 patients. However, in the United States, the practice is to place patients in single rooms where possible, with a maximum number of 2 patients per room.[9] Indeed, the 2006 American Institute of Architects (AIA) guidelines now mandate single rooms for all patients in new hospitals.[15] Consequently, whereas European hospitals frequently contain multiple-bed wards, their counterparts in the United States are composed largely of single- and 2-patient rooms. However, the AIA's requirement for single-bed patient rooms in US hospitals does not extend to critical care facilities, where multiple-bed wards are permitted (and indeed are the norm).

EVIDENCE FOR AERIAL DISSEMINATION

Before focusing on ventilation systems, it is worth considering the evidence regarding the airborne transmission of infection in hospitals. A full discussion of this topic is beyond the scope of this review, however, and thus we give only a brief overview of the evidence for the aerial dissemination of pathogens within the ward environment. This overview is restricted to those infections that normally are not considered airborne in nature and thus does not cover TB, legionnaire's disease, or pulmonary aspergillosis, which are already accepted as being transmitted by the airborne route.

There is a large body of evidence supporting the view that staphylococci are frequently disseminated by the aerial route in the clinical environment. Contaminated clothing and bedding of colonized patients release *Staphylococcus aureus* into the air when disturbed.[13,16,17] During bed-making in particular, staphylococci-bearing particles are liberated into the air and deposited on surfaces within the environment.[12,13,18] This process was well illustrated by Rutala et al,[19] who investigated a methicillin-resistant *S aureus* (MRSA) outbreak in a burn unit and found that MRSA accounted for 16% of all bacterial isolates sampled from the air and 31% of the isolates cultured from elevated surfaces. Because health care personnel or patients are unlikely to touch elevated surfaces, the presence of MRSA isolates on these surfaces suggests that staphylococci are frequently transported through the air. Although the clinical relevance of staphylococcal contamination is not fully known, a correlation between environmental contamination and patient infection/colonization has been noted by several researchers. Wilson et al[20] observed a strong correlation between the presence of MRSA-colonized or -infected patients and air samples yielding MRSA in an ICU. Boyce et al[21] found a similar correlation, with environmental contamination occurring in the rooms of 73% of MRSA-infected patients. In another study, Shiomori et al,[13] sampling the environment around MRSA-colonized or -infected patients under normal conditions, found an average of 4.7 cfu/m^3 MRSA-carrying particles in the air near infected patients; however during bed making, this figure increased to 116 cfu/m^3, confirming that this activity results in considerable aerosolization of staphylococci. Collectively, these findings suggest that MRSA-colonized or -infected patients readily contaminate their surroundings by aerial dissemination. Although the clinical relevance of this finding is incompletely understood, it may be that the resulting environmental contamination both increases the spread of the MRSA infection and prolongs any outbreaks that occur.

S aureus often colonizes the anterior nares, with about 20% of healthy people having persistent nasal colonization and about 60% displaying intermittent carriage.[22] It appears that the nose acts as a reservoir, which then supports colonization of the skin surface of most carriers; eradication of staphylococci from the nose is generally accompanied by eliminating *S aureus* from the other colonized body sites.[23] Given that humans liberate approximately 3×10^8 squama per day[24] and that each skin squame may carry > 100 bacteria,[25] there is a strong likelihood that the nares of susceptible adults can become colonized with *S aureus* simply by inhaling particles from the air,[26] and that this is likely to be a dose-related response.[27] Indeed, this has led one commentator to conclude that ''the principal mode of transmission is via transiently contaminated hands of hospital personnel…airborne transmission seems important in the acquisition of nasal carriage.''[28]

Hospital ventilation systems also have been implicated in MRSA outbreaks. Kumari et al[29] presented

evidence of patients acquiring MRSA as a result of periodic dispersion of MRSA-contaminated dusts from air grills. Cleaning the grills and ensuring continuous operation of the ventilation system prevented further outbreaks of MRSA infection. Wagenvoort et al[30] found MRSA isolates on ventilation grills in an orthopedic ward, and Cotterill et al[31] identified colonies of MRSA in the exhaust air from an isolation room as the source of an outbreak in an intensive care unit; the MRSA bacteria were reentering the unit through an open window.

Another important nosocomial pathogen for which there is growing evidence of aerial dissemination is *Acinetobacter*. *Acinetobacter* spp are the only gram-negative bacteria that form part of the normal skin microflora, with colonization in 25% to 43% of healthy people.[32] Unlike most gram-negative bacteria, they are particularly hardy and survive well in the environment.[32-34] Consequently, *Acinetobacter* spp can be readily disseminated on skin squama in a manner similar to *S aureus*.

Numerous studies have implicated the aerial dissemination of *Acinetobacter* spp bacteria in the transmission of infection. Allen and Green[35] were the first to suggest airborne dissemination of *Acinetobacter*-carrying particles. Investigating an outbreak of multiply antibiotic-resistant *Acinetobacter anitratus* in an intensive care unit (ICU), a medical ward, and 3 neurosurgical wards, these investigators cultured the outbreak organism from 16 of 82 settle plates, leading them to conclude that widespread aerial dissemination of *Acinetobacter* spp was occurring. Based on results of Allen and Green and of their own study, Das et al[36] suggested that movement of heavily contaminated bed curtains could promote the airborne spread of *Acinetobacter* spp Further evidence of the aerial dissemination of *Acinetobacter* spp came from a study in Hong Kong, in which Houang et al[37] placed 70 settle plates in an ICU and 120 (in total) in 4 surgical wards. Remarkably, 96% of plates in the ICU and 89% in the surgical wards were culture-positive, demonstrating widespread airborne dispersal. In a Danish study, Gerner-Smidt[38] recovered an outbreak of strain *Acinetobacter calcoaceticus* subspecies *anitratus* from the air in an ICU using both settle plates and a slit sampler. Others also have shown that *Acinetobacter* spp can be readily cultured from hospital air.[39-42]

Some of the strongest evidence regarding the airborne spread of *Acinetobacter* spp comes from outbreaks of *Acinetobacter baumannii* in 3 Dutch hospitals (2 of which experienced outbreaks despite isolation precautions). Bernards et al[14] found strong evidence of *Acinetobacter* transmission by the airborne route. In the hospitals that experienced outbreaks, the source patients were isolated in nonpressurized rooms, whereas in the other hospital, the infectious patient was isolated in a negatively pressurized room. In the hospitals with outbreaks, the settle plates, located inside and outside the isolation rooms, grew the outbreak strain, whereas in the third hospital, plates placed in the same location proved to be culture-negative. Thus, Bernards et al surmised that airborne transmission was occurring.

Clostridium difficile–associated disease (CDAD), a major problem on elderly care wards, is known to be associated with environmental contamination.[43-45] It has been postulated that environmental contamination might result from aerial dissemination of *C difficile* spores, which can survive on inanimate surfaces for several months.[46,47] Evidence supporting this supposition comes from a 22-month surveillance study in which air vents and high horizontal surfaces were found to be contaminated with *C difficile*, suggesting the aerial dissemination of isolates.[45,48] Moreover, numerous studies have found *C difficile* isolates on patients' bedding.[47,49,50] Given that bed-making is known to liberate large numbers of bacterial-carrying particles into the air,[51-53] the presence of *C difficile* on patient's bed linen suggests that *C difficile* spores or vegetative cells may be disseminated into the air by this route. Indeed, in a recent study on an elderly care ward,[54] we managed to culture *C difficile* from the air on 23 separate occasions over a 2-day period, with counts ranging from 53 to 426 cfu/m^3 of air, suggesting the presence of a significant source within the ward during the sampling period.

Although the foregoing discussion is far from exhaustive, it illustrates the fact that in hospital wards, pathogenic microorganisms are frequently liberated into the air in relatively large quantities. If not ventilated from the ward space, these airborne pathogens will cause widespread environmental contamination as they settle on surfaces within the ward, thus seeding potential reservoirs of infection. Given this situation, there is reason to believe that if used appropriately, ward ventilation may help control the spread of some nosocomial infections.

VENTILATION GUIDELINES IN THE UNITED KINGDOM AND UNITED STATES

Table 1 summarizes the ventilation and comfort standards for general ward spaces and ICUs as promulgated by the various regulatory bodies in the United States and United Kingdom. In the United States, the AIA guidelines regulate the design of health care facilities.[9] The AIA guidelines are supplemented by the guidelines of the American Society of Heating, Refrigeration, and Air-Conditioning Engineers (ASHRAE).[10] In the United Kingdom, HTM 2025, published by the Department of Health, is used to guide designers of health care facilities.[7]

Table 1. Comparison of the various guidelines governing the ventilation of general and intensive care ward spaces in the United Kingdom and the United States

Code	Country	Pressure Relationship	Minimum Outdoor Air Change Rate (AC/h)	Minimum Total Air Change Rate (AC/h)	Design Air Temperature (°C)	Design Relative Humidity (%)
Patient rooms/general wards						
AIA	United States	Neutral	2	6	21 to 24	Not specified
ASHRAE	United States	Neutral	2	6	21 to 24	30 to 60
HTM 2025	United Kingdom	Neutral	Not specified*	Not specified†	20 to 22	40 to 60
Intensive care wards						
AIA	United States	Neutral	2	6	21 to 24	30 to 60
ASHRAE	United States	Neutral	2	6	21 to 24	30 to 60
HTM 2025	United Kingdom	Neutral	Not specified*	Not specified†	20 to 22	40 to 60

*Minimum outdoor air (ie, fresh air) rate of 8 l/s per person specified.
†100% outdoor air encouraged.

From Table 1, it can be seen that the guidelines in the United States are more prescriptive than those in the United Kingdom, the main difference being that the AIA guidelines specify minimum ventilation rates (ie, air change rates), whereas HTM 2025 does not (other than requiring a minimum fresh air rate of 8 l/s per person). In addition, the AIA guidelines permit recirculation of ward air, whereas HTM 2025 strongly discourages (although does not completely outlaw) the use of recirculation systems. With regard to the comfort conditions, Table 1 shows that the internal design requirements are similar in the United Kingdom and United States.

In the United States, the air supplied to patients in general wards must be first prefiltered (minimum efficiency reporting value [MERV] 7, 30% dust spot efficiency), and then filtered to a MERV 14 or 15 standard (90% to 95% dust spot efficiency) before delivery to the ward space.[10] This standard of filtration ensures 85% to 95% arrestance efficiency for 0.3 to 1.0 μm particles and > 90% efficiency for > 1.0 μm particles. Given that skin squama are generally 4 to 25 μm in size, this level of filtration should ensure that the air supplied to the ward space is relatively clean, despite the fact that a large proportion of this air may be recirculated. In the United Kingdom, where ward mechanical ventilation systems tend to be full fresh air, HTM 2025 is somewhat vague on the subject of filtration. It simply specifies EU5 filters (50% dust spot efficiency) for ''general applications where décor protection is not critical'' and EU6 filters (70% dust spot efficiency) for general applications where décor protection is particularly important, making no reference to clinical requirements.

DRIVERS

Analysis of HTM 2025 reveals that with respect to the ventilation of general ward spaces, the guidance notes are driven by comfort and economic issues rather than by clinical considerations. As long as reasonable patient comfort conditions are maintained, the regulations are not concerned about whether ventilation is achieved by natural or mechanical means. Indeed, HTM 2025 actively encourages natural ventilation, although it is very vague as to how this should be achieved in practice. This reliance on natural ventilation may explain in part why HTM 2025 does not specify minimum ventilation rates. With regard to mechanical ventilation, HTM 2025 simply states that ''where mechanical supply systems are required, the fresh air should be tempered and filtered before being delivered to the space, to avoid discomfort,'' and with regard to air-conditioning, that ''air-conditioning is only required in a very small number of areas within health care buildings; and due to the capital and running cost implications, its inclusion should be kept to a minimum.''

Although HTM 2025 actively promotes the use of natural ventilation, it does acknowledge that in larger health care facilities, where internal spaces are greater (ie, deeper) than 6 m from a facade, mechanical ventilation generally will be required. In such situations, it recommends using a 100% fresh air system, presumably to avoid the recirculation of airborne pathogens. Such a system allows the use of a lower standard of filtration compared with similar ventilation systems in the United States.

The regulations in the United States take a different approach to those in the United Kingdom, specifying minimum fresh air and total ventilation rates for different applications. This is due primarily to the fact that the United States experiences climatic extremes that preclude the use of natural ventilation for much of the year. Consequently, mechanical ventilation and air-conditioning systems are used much more widely in the United States than in the United Kingdom. The provision for air recirculation in the AIA guidelines primarily reflects the desire to reduce energy costs while

still maintaining a comfortable internal room condition. This in turn explains why the filtration standards are much higher in the United States than in the United Kingdom. Clearly, the drafters of the AIA guidelines recognized the infection risks associated with recirculating unfiltered air. Notwithstanding this, to allow for greater flexibility, mechanical ventilation systems that use 100% fresh air are frequently installed in US hospitals, thus permitting the use of patient rooms as airborne infection isolation rooms.

Along with comfort and economic issues, infection control appears to be a driving force behind policy decisions in the United States. In the 2001 AIA guidelines, the total air change rate requirement for patient rooms was increased from 2 air changes per hour (AC/h) to 6 AC/h to improve patient comfort.[6] The increased total ventilation rate also provided a measure of protection against patients with undiagnosed TB.[55] Unlike HTM 2025, which puts little emphasis on room humidity, the ASHRAE guidelines address the effects of high humidity on the proliferation of pathogens within the clinical environment, specifying the maintenance of ward relative humidity at 30% to 60%.[10] Achieving this level necessitates the use of air-conditioning in many parts of the United States during the summer months, increasing energy costs. It is noteworthy that neither the US or the UK guidelines attempt to specify airflow patterns within ward spaces, but instead rely on good air mixing to promote a good dilution affect.

PATIENT DENSITY AND ACTIVITY

One potential weakness of simply quoting required air change rates is that this approach takes no account of patient density—the ventilation rate is determined solely by the room volume, rather than the number of occupants. In reality, as ward occupancy levels increase, bioaerosol production within the space also increases. Any increase in the number of beds in a ward space will be accompanied by a corresponding increase in the number of nursing staff and visitors, all of whom will liberate microorganisms into the air. Indeed, even a modest increase in the number of patients may result in a substantial increase in bioaerosol production. Thus, if a ventilation system is required to control the bioaerosol level in a ward space, then it may be desirable to link its specification to ward occupancy levels in some way. In the United States, the AIA guidelines do this by specifying the size of rooms and strictly limiting the number of patients per room.[15]

In reality, bioaerosol production on wards is not constant, but varies greatly throughout the day with changes in activity level. In a recent study conducted in a respiratory ward, we found that such activities as bed-making, patient washing, and ward rounds produced significant increases in the number of airborne particles > 3 μm in size liberated into the air.[53] This finding is intuitive, given that large numbers of skin squama are likely to be liberated into the air during these activities.

INFLUENCES ON PATHOGEN TRANSPORT AND REMOVAL

Both the UK and US guidelines assume the use of dilution ventilation when ventilating general ward spaces. Such a strategy relies on good air mixing within the room space and generally is achieved by supplying clean filtered air in through diffusers in the ceiling and extracting contaminated air out through grills also located in the ceiling. With this type of ventilation system and full air mixing, the steady-state contaminant level, C_e, achieved in the ward space can be calculated as

$$C_e = \frac{q_c}{Q_v}, \tag{1}$$

where q_c is the generation rate of biological contaminants in the room space (cfu/s) and Q_v is the volume flow rate of the ventilation air (m^3/s). This equation shows that the greater the ventilation air flow rate, the lower the contaminant concentration level in the room air, and the greater the air change rate, the shorter the average residence time of bioaerosol particles in the room space. At a ventilation rate of 2 AC/h, the average particle residence time is 30 minutes; increasing the rate to 6 AC/h decreases the average residence time to 10 minutes.

PARTICLE SIZE DISTRIBUTION

The ability of any given ventilation system to remove particles from a room space does not depend solely on the air change rate. In reality, air in ventilated rooms usually is far from fully mixed,[56] and the concentration of bioaerosols depends on the location of the bioaerosol source, the local airflow patterns, and the size distribution of the particles. Very small particles fall through the air very slowly and thus are much more likely to be removed by the ventilation system, whereas large particles are much more likely to remain in the room space, albeit displaced somewhat by the room air currents before settling out. The terminal velocity of particles falling through air can be calculated using Stokes' law,

$$V_t = \frac{\rho_p d^2 g}{18\eta} C_c, \tag{2}$$

where ρ_p is the density of the particle (kg/m^3), d is the particle diameter (m), g is the acceleration due to gravity

Table 2. Terminal velocity of falling particles, assuming a particle density of 1000 kg/m^3

Particle Diameter (μm)	Terminal Velocity (mm/s)	Time Required to Fall 2 m (minutes)
1	0.036	932.1
2	0.133	251.1
4	0.504	66.2
8	2.001	16.7
16	7.791	4.2
32	31.886	1.0

(9.81 m/s^2), η is the viscosity of air (1.78×10^{-5} kg/ms), and C_c is the Cunningham slip-correction factor.

To highlight the significance of eq (2), Table 2 presents information demonstrating how the terminal velocity and duration of fall of a typical particle varies with its diameter. The table shows that small changes in diameter greatly influence settling velocity, with particles < 4 μm taking hours to fall 2 m in a still room, compared with particles > 4 μm, which take minutes to fall the same distance. Although air flow patterns in room spaces can be highly complex, it is possible to make some general statements about the fate of different-sized bioaerosol particles liberated into the air. Because of their small mass and very slow terminal velocity, most particles < 5 μm are likely to be extracted from the room space, although some eventually may be deposited on surfaces after having first been transported some distance. In comparison, the fate of larger particles is somewhat less clear. Some will be removed completely from the room space by the ventilation system, whereas many others (usually the largest) will be deposited on various surfaces throughout the room space. Therefore, particle size has a considerable affect on the eventual fate of microorganisms liberated into the air.[57] Microorganisms aerosolized in respiratory droplet nuclei are most likely to be extracted from the ward space by the ventilation air, whereas those released on larger skin squama are much more likely to result in environmental contamination of room surfaces.

VENTILATION STRATEGY

Notably, the guidelines for ventilating general ward spaces in both the United Kingdom and the United States make no attempt to specify airflow patterns and assume that dilution ventilation will be used. This situation may be due in part to the complexity of airflow patterns in rooms, as alluded to earlier; however, it is perhaps worth considering the affect of air flow direction on bioaerosol concentration generated within a ward space. Consequently, we carried out a short CFD study to explore this affect by simulating 3 different ventilation strategies in an empty 32-m^3 room, as shown in Figure 1. The study was done using Fluent 6.2 CFD software (ANSYS, Canonsburg, PA) with an unstructured tetrahedral grid containing approximately 540,000 cells. A standard k-ε turbulence model with enhanced wall treatment was used, and a no-slip condition was applied at the walls. The model was treated as isothermal in all cases. The 3 ventilation regimes were as defined in Table 3. In cases 1 and 2, the supply air diffuser was modeled by a series of parabolic velocity profiles representing the grill louvers, with the air entering at a downward angle of 45 degrees in case 1 and entering horizontally in case 2. In case 3, the supply air entered at a downward angle of 45 degrees from the sides of the ceiling located box, to represent a 4-way diffuser. In all cases, the total air flow rate was set to be equivalent to 6 AC/h, and a zero pressure condition was defined on the extract diffuser boundary.

Bioaerosols were modeled using a transported scalar to represent the concentration of airborne particles. This assumes that all of the bioaerosol particles remain suspended in the air, with none settling out. Although this assumption may not be true for larger skin squama, it is a good approximation for smaller particles

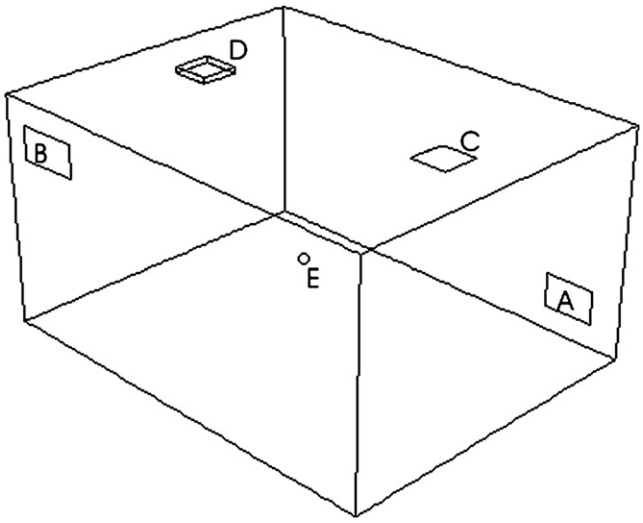

Fig 1. Geometry of the room showing the location of ventilation grilles (A to D) and the bioaerosol source (E).

Table 3. Ventilation regimes simulated using the CFD model

Ventilation Regime	Supply Diffuser Location	Extract Diffuser Location
1 (low–high)	A	B
2 (high–low)	B	A
3 (ceiling)	D	C

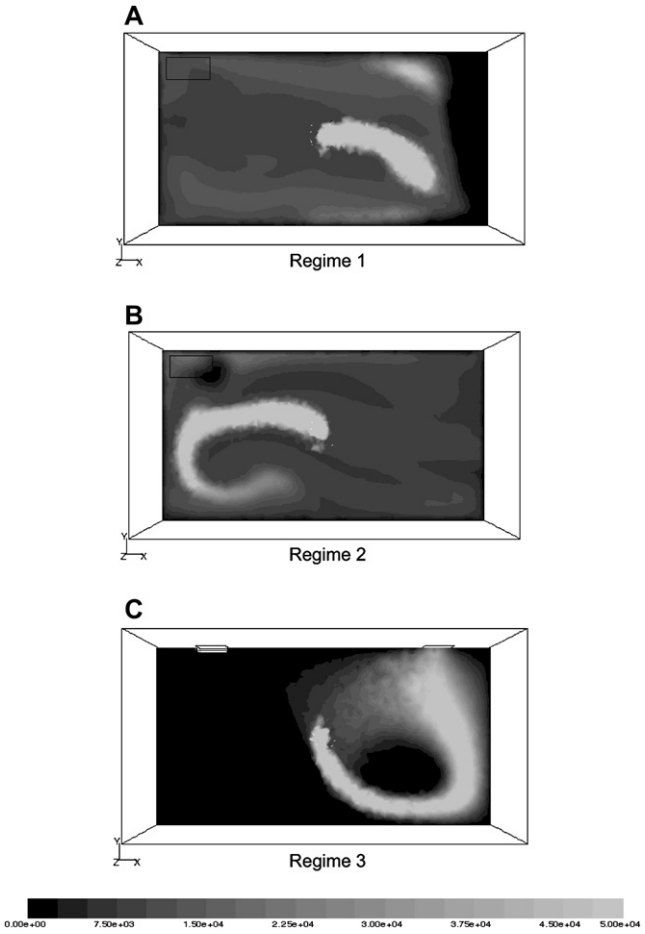

Fig 2. Bioaerosol concentration contour plots on an *x*–*y* vertical plane located centrally in the room and facing diffuser A. *A*, Regime 1. *B*, Regime 2, *C*, Regime 3. Concentration in cfu/m^3.

(ie, < 10 μm). The bioaerosols are assumed to enter the space at a constant rate over a small volume (0.1 m^3) located in the center of the room (point E in Fig 1), representing a point source due to an infectious patient. This is modeled through a constant volumetric source term and a momentum term of 0.1 N/m to represent the inertia of the particles on their release.

We solved the model just described using second-order discretization and a segregated implicit solver to find steady-state simulations for the 3 ventilation cases. Convergence was good in all 3 models, with a mass imbalance of $< 0.1\%$ in the final solutions. Results from the CFD simulations are presented in Figure 2 and Table 4. Figure 2 shows bioaerosol concentration contours plotted on a vertical plane through the center of the room looking toward diffuser A. For clarity, the maximum contour plotted is 50,000, although the highest concentration close to the source is of the order of 1,700,000. Table 4 presents the volume average bioaerosol concentration throughout the entire room, calculated for each regime using the postprocessing tools in Fluent.

Table 4. Volume-averaged bioaerosol concentration calculated from CFD simulation results for 3 ventilation regimes

Ventilation Regime	Volume Average Concentration (cfu/m^3)
1 (low–high)	12,487
2 (high–low)	10,601
3 (ceiling)	2467

Both the average data and the contour plots show significant variation between the 3 ventilation regimes. The lowest average value is seen in case 3, in which the supply and extract are located in the ceiling. Cases 1 and 2 have concentrations of similar orders of magnitude, up to 5 times greater than that of case 3. The reason for this difference is apparent from the contour plots. In cases 1 and 2, the airflow distributes the contaminant across the plane, and in fact draws the plume emitted from the source initially toward the supply diffuser side of the room. This means that the airflow promotes mixing in the room, following the classic theory of the dilution effect. But the contour plots for case 3 reveal an airflow pattern such that the contaminant is very effectively removed from the room before mixing occurs. Thus, there is a high bioaerosol concentration between the source location and the extract grill, but little contamination distributed across the rest of the plotted plane.

Care should be taken to distinguish between piston ventilation (as shown by regime 1 in the foregoing CFD model) and displacement ventilation systems. The latter relies on the buoyancy effects caused when cool air supplied at low level and is warmed when coming into contact with room occupants. Displacement ventilation systems have been used successfully in many applications, but their suitability in clinical applications is unclear. This is because in the ward environment, microorganisms often are projected into the air with some force, either through respiratory expulsions (eg, coughing)[58] or as a result of activities (eg, bedmaking).[51-53] Consequently, bioaerosols rapidly become decoupled from the buoyancy-driven plumes that surround room occupants when displacement ventilation is used. In a recent experimental study, Qian et al[59] found that with displacement ventilation, when patients cough, the exhaled jets thus formed penetrated long distances, resulting in ''trapped'' regions of high concentrations of exhaled droplet nuclei that could not be not rapidly dissipated by the ventilation air, but with dilution (ie, mixing) ventilation, the exhaled

jets penetrated only a short distance and were quickly diluted by the ventilation air. The investigators thus concluded that the type of ventilation strongly influenced bed spacing, with displacement ventilation necessitating spacing the beds further apart compared with when dilution ventilation is used. In addition, Zhao et al,[57] in a theoretical CFD study of an empty room, found that whereas displacement ventilation generally reduced particle deposition on surfaces, it greatly increased the number of particles suspended in the room air, particularly larger particles (ie, $>$ 10 μm). They concluded from this somewhat surprising finding that whereas the unidirectional air flow enabled smaller particles to escape the space, the larger particles attempted to settle in the opposite direction and thus remained suspended in the air for long periods.

These published results, together with the CFD model presented in this study, serve to further highlight the complexity of air flow in rooms and its dependence on the local room design. The findings from studies on displacement ventilation suggest that this ventilation method may not be well suited to general ward spaces, and that dilution ventilation can better control the spread of infection. Although in the model presented herein, 3 different dilution ventilation regimes are considered, drawing general conclusions about the most appropriate design for a hospital environment is difficult. Nonetheless, the results of the present study and those from simulations of a TB ward presented in a previous study demonstrate that the ventilation system within a single room space can have a significant affect on the distribution of airborne infectious material, and thus on the risk of cross-infection.[60] This suggests that although it may not be appropriate for inclusion in guidelines, ventilation system designers should seriously consider using CFD and other simulation tools to optimize ventilation design to minimize infection risk, and that further studies are needed to properly understand the influence of the airflow patterns.

DISCUSSION

From the foregoing discussion, it is clear that ventilation systems for general wards and patient rooms are specified using criteria that differ little from those used for nonclinical spaces. The guidelines in both the United Kingdom and the United States avoid any discussion of the risks posed by airborne microorganisms, but focus on providing a comfortable environment. This is understandable, given that patient comfort is of great importance and that the clinical risk posed by many airborne pathogens is unclear. Nonetheless, there is growing evidence that the aerial dispersion of some nosocomial pathogens is seeding widespread environmental contamination that may be promoting infection in immunocompromised patients.[11,12,21] *Acinetobacter* spp in particular appears conform to this model, with numerous outbreaks attributed in to its aerial dissemination.[61] If the aerial dissemination of microbes is indeed contributing to overall levels of infection in any way, then ward ventilation becomes very important, because ventilation design has a considerable affect on the eventual fate of airborne microorganisms. It may be possible to greatly reduce environmental contamination and thus minimize HAI through improved ventilation.

Given the considerable body of evidence indicating that aerial dissemination of skin squama from such activities as bed-making has the potential to cause widespread environmental contamination, air flow patterns within ward spaces would seem to be an issue of some importance. This is particularly true if it is important to ensure that clinically sensitive surfaces remain free of microbial contamination. Toward this end, piston-type ventilation may offer some benefits over conventional dilution systems. In contrast, displacement ventilation that relies on natural buoyancy plumes appears to offer only modest benefits, because the air velocities associated with this type of ventilation generally are very low. Indeed, there is some evidence that displacement ventilation is rather poor at removing larger ($>$ 10 μm) particles from the air; [57] however, because of the limited number of studies undertaken, these observations cannot be considered definitive for all situations.

CFD modeling is a powerful tool for investigating ventilation strategies. The models used in this work have yielded useful data on the spread of an idealized source of contamination in an isothermal situation. This technique possibly may be further extended to include more detailed models of the physics of contamination transport—for example, the temperature and relative humidity of the air, to account for buoyancy of particles and for changes in particle size due to evaporation. But although more sophisticated models may be developed, producing accurate simulations is impossible without the input of good data describing the particle source and the size range and volume of the particles produced. In particular, it is important to allow for heat sources within the room space, because these have been shown to significantly influence both air flow and thermal comfort.[62-64]

Although there is strong evidence that good ward ventilation provides health benefits, because of the complexity of the mechanisms involved, the level of ward ventilation required to prevent HAI is not known. Indeed, in a recent study, Li et al[65] concluded that the ''strong and sufficient evidence of the association between ventilation, the control of airflow direction in buildings, and the transmission and spread of

infectious diseases supports the use of negatively pressurized isolation rooms for patients with these diseases in hospitals, in addition to the use of other engineering control methods. However, the lack of sufficient data on the specification and quantification of the minimum ventilation requirements in hospitals, schools and offices in relation to the spread of airborne infectious diseases, suggest the existence of a knowledge gap. Our study reveals a strong need for a multidisciplinary study in investigating disease outbreaks, and the impact of indoor air environments on the spread of airborne infectious diseases.'' This statement is very apt, because it reflects both our opinion and the frustrations of other researchers in the field. There is a clear knowledge gap regarding the extent to which airborne pathogens, respirable or otherwise, contribute to infection. Furthermore, there is a lack of good-quality data from which to make decisions regarding the minimum ventilation rates required to prevent infection. Good data will help those drafting future guidelines for ventilation designers reach firm conclusions.

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References

1. Blowers R, Mason GA, Wallace KR, Walton M. Control of wound infection in a thoracic surgery unit. Lancet 1955;269:786-94.
2. Shooter RA, Taylor GW, Ellis G, Ross JP. Post-operative wound infection. Surg Gynecol Obstetr 1956;103:257-62.
3. Leung M, Chan AH. Control and management of hospital indoor air quality. Med Sci Monit 2006;12:SR17-23.
4. Smyth ET, Humphreys H, Stacey A, Taylor EW, Hoffman P, Bannister G. Survey of operating theatre ventilation facilities for minimally invasive surgery in Great Britain and Northern Ireland: current practice and considerations for the future. J Hosp Infect 2005;61:112-22.
5. Chow TT, Yang XY. Ventilation performance in operating theatres against airborne infection: review of research activities and practical guidance. J Hosp Infect 2004;56:85-92.
6. Ninomura P, Bartley J. New ventilation guidelines for health-care facilities. ASHRAE J 2001;43:29-33.
7. National Health Service. Design considerations: ventilation in healthcare premises. Health technical memorandum 2025. London: National Health Service Estates; 1994.
8. Sehulster LM, Chinn RYW, Arduino MJ, Carpender J, Donlan R, Ashford D, et al. Guidelines for environmental infection control in healthcare facilities: recommendations from CDC and Healthcare Infection Control Practices Advisory Committe (HICPAC). Atlanta (GA): US Department of Health and Human Services, Centers for Disease Control and Prevention; 2003.
9. American Institute of Architects. Guidelines for design and construction of hospital and health care facilities. Washington, DC: American Institution of Architects; 2001.
10. American Society of Heating, Refrigeration and Air-Conditioning Engineers. HVAC design manual for hospitals and clinics. Atlanta (GA): American Society of Heating, Refrigeration and Air-Conditioning Engineers; 2003.
11. Beggs CB, Noakes CJ, Shepherd SJ, Kerr KG, Sleigh PA, Banfield K. The influence of nurse cohorting on hand hygiene effectiveness. Am J Infect Control 2006;34:621-6.
12. Shiomori T, Miyamoto H, Makishima K. Significance of airborne transmission of methicillin-resistant *Staphylococcus aureus* in an otolaryngology–head and neck surgery unit. Arch Otolaryngol Head Neck Surg 2001;127:644-8.
13. Shiomori T, Miyamoto H, Makishima K, Yoshida M, Fujiyoshi T, Udaka T, Inaba T, Hiraki N. Evaluation of bedmaking-related airborne and surface methicillin-resistant *Staphylococcus aureus* contamination. J Hosp Infect 2002;50:30-5.
14. Bernards AT, Frenay HM, Lim BT, Hendriks WD, Dijkshoorn L, van Boven CP. Methicillin-resistant *Staphylococcus aureus* and *Acinetobacter baumannii*: an unexpected difference in epidemiologic behavior. Am J Infect Control 1998;26:544-51.
15. American Institution of Architects. Guidelines for design and construction of health care facilities. Washington, DC: American Institution of Architects; 2006.
16. Noble WC, Davies RR. Studies on the dispersal of staphylococci. J Clin Pathol 1965;18:16-9.
17. Solberg CO. A study of carriers of *Staphylococcus aureus*. Acta Med Scand 1965;178(suppl):436.
18. Noble WC. The dispersal of staphylococci in hospital wards. J Clin Pathol 1962;15:552-8.
19. Rutala WA, Katz EB, Sherertz RJ, Sarubbi FA Jr. Environmental study of a methicillin-resistant *Staphylococcus aureus* epidemic in a burn unit. J Clin Microbiol 1983;18:683-8.
20. Wilson RD, Huang SJ, McLean AS. The correlation between airborne methicillin-resistant *Staphylococcus aureus* with the presence of MRSA colonized patients in a general intensive care unit. Anaesth Intensive Care 2004;32:202-9.
21. Boyce JM, Potter-Bynoe G, Chenevert C, King T. Environmental contamination due to methicillin-resistant *Staphylococcus aureus*: possible infection control implications. Infect Control Hosp Epidemiol 1997; 18:622-7.
22. Kluytmans J, van Belkum A, Verbrugh H. Nasal carriage of *Staphylococcus aureus*: epidemiology, underlying mechanisms, and associated risks. Clin Microbiol Rev 1997;10:505-20.
23. Sands KEF, Goldmann DA. Epidemiology of *Staphylococcus* and group A streptococci. In: Bennett JV, Brachman PS, editors. Hospital infections. 4th ed. Philadelphia: Lippincott Raven; 1998.
24. Rhame FS. The inanimate environment. In: Bennett JV, Brachman PS, editors. Hospital infections. 4th ed. Philadelphia: Lippincott Raven; 1998.
25. Lundholm IM. Comparison of methods for quantitative determinations of airborne bacteria and evaluation of total viable counts. Appl Environ Microbiol 1982;44:179-83.
26. Noble WC. Dispersal of microorganisms from skin. In: Noble WC, editor. Microbiology of human skin. 2nd ed. London: Lloyd-Luke Ltd; 1981. p. 79-85.
27. Williams RE. Epidemiology of airborne staphylococcal infection. Bacteriol Rev 1966;30:660-74.
28. Solberg CO. Spread of *Staphylococcus aureus* in hospitals: causes and prevention. Scand J Infect Dis 2000;32:587-95.
29. Kumari DN, Haji TC, Keer V, Hawkey PM, Duncanson V, Flower E. Ventilation grilles as a potential source of methicillin-resistant *Staphylococcus aureus* causing an outbreak in an orthopaedic ward at a district general hospital. J Hosp Infect 1998;39:127-33.
30. Wagenvoort JH, Davies BI, Westermann EJ, Werink TJ, Toenbreker HM. MRSA from air-exhaust channels. Lancet 1993;341:840-1.
31. Cotterill S, Evans R, Fraise AP. An unusual source for an outbreak of methicillin-resistant *Staphylococcus aureus*. J Hosp Infect 1996;32: 207-16.
32. Jawad A, Seifert H, Snelling AM, Heritage J, Hawkey PM. Survival of *Acinetobacter baumannii* on dry surfaces: comparison of outbreak and sporadic isolates. J Clin Microbiol 1998;36:1938-41.
33. Wagenvoort JHT, Joosten EJAJ. An outbreak of *Acinetobacter baumannii* that mimics MRSA in its environmental longevity. J Hosp Infect 2002; 52:226-7.

34. Jawad A, Snelling AM, Heritage J, Hawkey PM. Exceptional desiccation tolerance of *Acinetobacter radioresistens*. J Hosp Infect 1998;39: 235-40.
35. Allen KD, Green HT. Hospital outbreak of multi-resistant *Acinetobacter anitratus*: an airborne mode of spread? J Hosp Infect 1987; 9:110-9.
36. Das I, Lambert P, Hill D, Noy M, Bion J, Elliott T. Carbapenem-resistant *Acinetobacter* and role of curtains in an outbreak in intensive care units. J Hosp Infect 2002;50:110-4.
37. Houang ET, Chu YW, Leung CM, Chu KY, Berlau J, Ng KC, et al. Epidemiology and infection control implications of *Acinetobacter* spp in Hong Kong. J Clin Microbiol 2001;39:228-34.
38. Gerner-Smidt P. Endemic occurrence of *Acinetobacter calcoaceticus biovar anitratus* in an intensive care unit. J Hosp Infect 1987;10: 265-72.
39. Thornton T, Fletcher LA, Beggs CB, Elliott MW, Kerr KG. Airborne microflora in a respiratory ward. Presented at the ASHRAE IAQ Conference, Tampa, FL, March 15–17, 2004.
40. Obbard JP, Fang LS. Airborne concentrations of bacteria in a hospital environment in Singapore. Water, Air, and Soil Poll 2003;144:333-41.
41. Augustowska M, Dutkiewiez J. Variability of airborne microflora in a hospital ward within a period of one year. Ann Agric Environ Med 2006;13:99-106.
42. Kerr KG, Beggs CB, Dean SG, Thornton J, Donnelly JK, Todd NJ, et al. Air ionisation and colonization/infection with methicillin-resistant *Staphylococcus aureus* and *Acinetobacter* species in an intensive care unit. Intensive Care Med 2006;32:315-7.
43. Malamou-Ladas H, O'Farrell S, Nash JQ, Tabaqchali S. Isolation of *Clostridium difficile* from patients and the environment of hospital wards. J Clin Pathol 1983;36:88-92.
44. Hota B. Contamination, disinfection, and cross-colonization: are hospital surfaces reservoirs for nosocomial infection? Clin Infect Dis 2004;39:1182-9.
45. Fawley WN, Wilcox MH. Molecular epidemiology of endemic *Clostridium difficile* infection. Epidemiol Infect 2001;126:343-50.
46. Samore MH, Venkataraman L, DeGirolami PC, Arbeit RD, Karchmer AW. Clinical and molecular epidemiology of sporadic and clustered cases of nosocomial *Clostridium difficile* diarrhea. Am J Med 1996; 100:32-40.
47. Fekety R, Kim KH, Batts DH, Browne RA, Cudmore MA, Silva J Jr, et al. Studies on the epidemiology of antibiotic-associated *Clostridium difficile* colitis. Am J Clin Nutr 1980;33(11 Suppl):2527-32.
48. Fawley WN, Freeman J, Wilcox MH. Evidence to support the existence of subgroups within the UK epidemic *Clostridium difficile* strain (PCR ribotype 1). J Hosp Infect 2003;54:74-7.
49. Kim KH, Fekety R, Batts DH, Brown D, Cudmore M, Silva J Jr, et al. Isolation of *Clostridium difficile* from the environment and contacts of patients with antibiotic-associated colitis. J Infect Dis 1981;143: 42-50.
50. Fekety R, Kim KH, Brown D, Batts DH, Cudmore M, Silva J Jr. Epidemiology of antibiotic-associated colitis; isolation of *Clostridium difficile* from the hospital environment. Am J Med 1981;70:906-8.
51. Greene VW, Vesley D, Bond RG, Michaelsen GS. Microbiological contamination of hospital air, II: qualitative studies. Appl Microbiol 1962; 10:567-71.
52. Greene VW, Vesley D, Bond RG, Michaelsen GS. Microbiological contamination of hospital air, I: quantitative studies. Appl Microbiol 1962; 10:561-6.
53. Roberts K, Hathway A, Fletcher LA, Beggs CB, Elliott MW, Sleigh PA. Bioaerosol production on a respiratory ward. Indoor Built Environ 2006;15:35-40.
54. Roberts K, Smith CF, Snelling AM, Kerr KG, Banfield K, Sleigh PA, et al. Aerial dissemination of *Clostridium difficile*. Submitted to the BMC Infectious Diseases; 2007.
55. Kosar D. The answer is 3. Engineering Systems 2002;60-70.
56. Noakes CJ, Beggs CB, Sleigh PA. Modelling the performance of upper room ultraviolet germicidal irradiation devices in ventilated rooms: comparison of analytical and CFD methods. Indoor Built Environ 2004;13:477-88.
57. Zhao B, Zhang Y, Li X, Yang X, Huang D. Comparison of indoor aerosol particle concentration and deposition in different ventilated rooms by numerical method. Building Environ 2004;39:1-8.
58. Tang JW, Li Y, Eames I, Chan PKS, Ridgway GL. Factors involved in the aerosol transmission of infection and control of ventilation in healthcare premises. J Hosp Infect 2006;64:100-14.
59. Qian H, Li Y, Nielsen PV, Hyldgaard CE, Wong TW, Chwang AT. Dispersion of exhaled droplet nuclei in a two-bed hospital ward with three different ventilation systems. Indoor Air 2006;16:111-28.
60. Noakes CJ, Sleigh PA, Escombe AR, Beggs CB. Use of CFD analysis in modifying a TB ward in Lima, Peru. Indoor Built Environ 2006;15:41-7.
61. Beggs CB, Kerr KG, Snelling AM, Sleigh PA. *Acinetobacter* spp and the clinical environment. Indoor Built Environ 2006;15:19-24.
62. Philips D, Sinclair RJ, Schulyer GD. Isolation room ventilation design case studies. Presented at the ASHRAE IAQ Conference, Tampa, FL, March 15–17, 2004.
63. Memarzadeh F, Jiang J. Methodology for minimizing risk from airborne organisms in hospital isolation room. ASHRAE Trans 2000;106: MN-00-11-02.
64. Memarzadeh F, Manning A. Thermal comfort, uniformity, and ventilation effectiveness in patient rooms: performance assessment using ventilation indices. ASHRAE Trans 2000;106: MN-00-11-03.
65. Li Y, Leung GM, Tang JW, Yang X, Chao CY, Lin JZ, et al. Role of ventilation in airborne transmission of infectious agents in the built environment: a multidisciplinary systematic review. Indoor Air 2007;17:2-18.