Room ventilation and the risk of airborne infection transmission in three health care settings within a large teaching hospital

Abstract

Background: Room ventilation is a key determinant of airborne disease transmission. Despite this, ventilation guidelines in hospitals are not founded on robust scientific evidence related to prevention of airborne transmission.

Methods: We sought to assess the effect of ventilation rates on influenza, tuberculosis (TB) and rhinovirus infection risk within three distinct rooms in a major urban hospital; a Lung Function Laboratory, Emergency Department (ED) Negative-pressure Isolation Room and an Outpatient Consultation Room were investigated. Air exchange rate measurements were performed in each room using CO_2 as a tracer. Gammaitoni and Nucci's model was employed to estimate infection risk.

Results: Current outdoor air exchange rates in the Lung Function Laboratory and ED Isolation Room limited infection risks to between 0.1 and 3.6%. Influenza risk for individuals entering an Outpatient Consultation Room after an infectious individual departed ranged from 3.6 to 20.7%, depending on the duration for which each person occupied the room.

Conclusions: Given the absence of definitive ventilation guidelines for hospitals, air exchange measurements combined with modelling afford a useful means of assessing, on a case-by-case basis, the suitability of room ventilation at preventing airborne disease transmission.

Introduction

Room ventilation acts to dilute and remove infectious airborne droplet nuclei (aerosols), and several epidemiologic investigations have underscored its significant role in determining airborne transmission of tuberculosis (TB), influenza, measles, rhinovirus and severe acute respiratory syndrome (SARS) in various indoor settings.[1-8] An extensive multi-disciplinary review conducted in the wake of the 2003 SARS epidemic[9] concluded that the relationship between ventilation and airborne transmission of diseases indoors was supported by strong and sufficient evidence. Despite this, the potential for transmission in hospitals has received little attention, and there is insufficient data to prescribe minimum ventilation rates.[9] Existing ventilation guidelines, of which there are several (summarised by Beggs et al.[10]), are therefore not founded on robust scientific evidence related to prevention of airborne transmission.

While some respiratory infections may be communicated by fomites or over short distances (less than a few metres) by large droplets (> approximately 20 μm) that subsequently result in direct contact with the respiratory tract , airborne transmission is likely to contribute to person-to-person spread over relatively long distances due the ability of small droplet nuclei (< 5μm) to remain suspended in air for extended periods.[11] In addition to clear evidence that the airborne route is a mode of spread for tuberculosis and measles, there is mounting evidence for its role in influenza and rhinovirus transmission.[7, 12-16]

Given that the significance of knowledge gaps outlined above was amplified by the 2009 H1N1 pandemic, we sought to determine the effect of ventilation on the risk of airborne infection posed by three common pathogens in a major teaching hospital. We aimed to

provide general information for use in evaluation of hospital design and infection control strategies and also to inform in-house patient management guidelines.

Methods

Setting

The Prince Charles Hospital (TPCH) is a major tertiary referral and university hospital located in south eastern Queensland, Australia. TPCH has 588 beds, including a large Pulmonology unit with 54 inpatient beds. The hospital had ~77,000 outpatient consultations and ~17,000 lung function tests in 2009/10, and a large Emergency Department delivered 43,000 occasions of service. Clinical services at TPCH were built in recent years, and the Outpatient Department and Lung Function Laboratory, both commissioned in 1999, underwent redevelopment in 2007. The Emergency Department was completed and commissioned in 2007. In 2009, there were 286 confirmed cases of H1N1 influenza diagnosed and managed at the hospital. An average of 7 cases of *Mycobacterium tuberculosis* are managed at TPCH each year within the Pulmonology Unit.

Our study targeted rooms within TPCH that encompassed a range of uses and were potential airborne transmission locations: (1) the Respiratory Investigation (Lung Function) Laboratory (169 m^3); (2) a Negative-pressure Isolation Room within the Emergency Department (ED) (24 m^3), and; (3) two separate but proximate Outpatient Consulting Rooms (Room A and Room B: 32 and 36 m^3, respectively). All rooms were mechanically ventilated. The Lung Function Laboratory and Outpatient Consulting Rooms were served by Air Handling Units (AHUs), with the latter sharing a common AHU. The ED Isolation Room was ventilated entirely by outdoor air drawn in by an exhaust fan.

Air exchange rate measurements

Air exchange rate measurements in the Lung Function Laboratory and ED Isolation Room were performed with the doors in their typical position; fully open and fully closed, respectively, whilst those in the two outpatient consulting rooms were conducted under both closed and open door conditions, representing a typical functioning outpatient clinic session. All measurements were performed when the rooms were unoccupied. Background concentrations of CO_2 were monitored by a Sable Systems CA-10 CO_2 analyser for at least 20 minutes. High purity (99.9%) CO_2 was then released and vigorously mixed with room air by two fans until concentrations stabilised. Approximate homogeneity of concentration was confirmed by measurements at a minimum of three points throughout each room prior to cessation of CO_2 release. A single sampling point was then sited at a central location. The decay of CO_2 was recorded every second until background concentrations were reached. Three repeat measurements were conducted in each room, and in the case of consulting rooms, for each door position. Eighteen air exchange measurements were performed.

The gradient of the line-of-best-fit through the natural logarithm of the background-corrected decay was recorded as the number of air changes per hour (ACH). The standard error of the line-fitting procedure was calculated.

For all rooms except the ED Isolation Room, the proportion of outdoor air in the total air volume supplied by their respective AHUs (i.e. the combination of outdoor and recirculated air) was determined by mass-balance of CO_2 concentrations measured in return, supply and outdoor air.[17] The precision of calculated values was estimated using the method described by Persily.[17]

Infection risk modelling

We employed Gammaitoni and Nucci's[18] (G-N) model to estimate airborne transmission risk. The G-N model is a variation of the traditional steady-state Wells-Riley[1] (W-R) model. Both models assume that an infectious person constantly generates a number of infectious quanta through time, with a quantum defined as the dose of airborne droplet nuclei required to cause infection in $1 - e^{-1}$, or 63% of susceptible persons. Unlike the W-R model, however, the G-N model is capable of incorporating non-steady-state quanta concentrations. Detailed discussions of each model's merits and underlying assumptions are provided by Beggs et al.[19] and Sze To and Chao.[20]

W-R model:

$$Risk = 1 - e^{-Iqpt/Q} \quad (1)$$

Where: I is the number of infectious source cases, q is the number of infectious quanta produced per source case (quanta/h), p is the average respiratory ventilation rate of susceptible persons (m^3/h), t is the duration of exposure (h) and Q is the volume of infection-free (i.e. outdoor) air supplied to the room (m^3/h).

G-N model (when initial quanta concentration is non-zero)[19]:

$$Risk = 1 - e^{\left(-\frac{pIq}{V}\frac{Nt+e^{-Nt}-1-\left(\frac{Nn_0}{qI}\right)e^{-Nt}+\left(\frac{Nn_0}{qI}\right)}{N^2}\right)} \quad (2)$$

Where: V is the volume of the room (m^3), N is the air change rate (i.e. Q/V) and n_0 is the total number of quanta in the room at $t = 0$.

We modelled three diseases spread by the airborne route that spanned a range of infectiousness and frequency of presentation at the study site; influenza, tuberculosis and rhinovirus. Quanta generation rates were 67, 12.7 and 5 quanta/hour, respectively, with these values chosen to represent relatively typical cases.[5,21] Although we did not explicitly model H1N1, the influenza quanta generation rate we used is within its suspected range.[22] We assumed that all susceptible individuals had a standard adult respiratory rate of 0.6 m^3/h.[5,19] The modelling approach and additional equations used are described in the appendix.

Based on typical patient occupancy times and patterns, two general scenarios were modelled for each airborne pathogen: (1) risk of infection for susceptible individuals occupying the Lung Function Laboratory with an infectious patient (exposure times ranging from 15 to 45 min), and; (2) risk of infection for susceptible individuals occupying the ED Isolation Room for between 30 min and 8 h immediately following the departure of an infectious individual who spent 30 min or more in the room. A third, more complex, situation was also modelled; (3) risk of infection for a susceptible individual occupying an Outpatient Consulting Room for up to 120 min after prior occupation by an infectious individual for each of 15, 60 and 120 min; which span the range of consultation times for a brief through to complex multi-disciplinary consultation. To mimic typical practice, a period of 5 minutes during which the door was open was incorporated into each scenario (i.e. between the departure of the infectious person and arrival of the susceptible individual). To best assess the capability of its ventilation system at preventing airborne transmission, the most infectious pathogen of those we investigated (influenza) was modelled in the outpatient consulting room.

Ethical Approval

The study was approved by the Human Research Ethics Committees of TPCH (HREC/09/QPCH/163) and Queensland University of Technology (0900001290). Individual patient consent was not required for this study, though signage explaining the purpose of the measurements was displayed to staff, patients and visitors.

Results

Table 1 summarises the results of air exchange and outdoor air proportion measurements. The proportion of outdoor air supplied to the Lung Function Laboratory by its AHU was approximately twice that of the Outpatient Consulting Rooms; the cause of which was traced to modification of the outdoor air intake dampers by technicians two years prior to our investigation. All outdoor air proportions were fixed and did not vary with season, reflecting the small seasonal temperature variation at the study location.

A significant finding was the effect of door position on air exchange rates in Outpatient Consulting Rooms A and B, which was most marked in the former and resulted in a near-doubling of air exchange compared to the closed door situation. We ascribe this to the presence of a large air return air vent in the corridor immediately adjacent to room A that promoted air movement out of the room, and that this effect was enhanced under open door conditions.

Lung Function Laboratory

Figures 1a and b show the effect of outdoor air exchange rate on the infection risk of susceptible individuals occupying the Lung Function Laboratory for 15 and 45 mins, respectively. For all scenarios, risk decreased rapidly with increasing air exchange. The current outdoor air exchange rate in the Lung Function Laboratory (4.9 ACH; shown as the

vertical dotted line in figures 1a and b) is relatively high and resulted in risks ranging from 0.1% (following 15 min exposure to rhinovirus) to 3.6% (following 45 min exposure to influenza).

Emergency Department (ED) Isolation Room

The high air exchange rate in the ED Isolation Room (23.8 ACH) resulted in steady-state quanta concentrations being achieved after approximately 15 mins, and these were consequently low; 0.12, 0.02 and 0.009 quanta/m^3 for influenza, TB and rhinovirus, respectively. The estimated time required to achieve a 99.9% reduction in quanta concentrations following the departure of an infectious individual is 18 min. The risk posed to an individual entering the room immediately after the departure of an infectious influenza case (i.e. worst-case scenario) and remaining there for 30 min or 8 h was 0.3%. The additional 7.5 hours of occupancy time in the latter case did not have a significant effect on risk, as no new sources of influenza quanta were present.

Outpatient (OPD) Consulting Rooms

Figure 2a shows the modelled influenza quanta concentration in Outpatient Consulting Room A during a consultation with an infectious individual for up to 120 min. Also shown are the decays in quanta concentration following departure of the individual after 15, 60 and 120 min, including an initial 5 min period with the door to the room open. Figure 2b shows the estimated risk of infection for a susceptible individual entering the room after each of these periods.

During a 15 min consultation with an infectious individual, there is insufficient time for the quanta concentration to reach its steady-state value (1.08 quanta/m^3). An initially sharp

decrease in quanta when the door is opened and the infectious individual departs is curtailed as the air exchange rate is lowered once the susceptible individual enters and the door is closed. As Figure 2b shows, the subsequent risk of infection to the susceptible individual ranges from 3.6 to 8.8% for 15 and 120 min consultations, respectively.

A similar pattern, albeit of greater magnitude, is repeated in the case of 60 and 120 min consultations with an infectious individual. In these scenarios, quanta concentrations approach their steady-state values at the conclusion of the consultation. When the room has previously been occupied for 60 min by an infectious person, the susceptible individual's estimated infection risk ranges from 8.1 to 18.5% for 15 and 120 min consultations, respectively. The equivalent range assuming prior occupation by an infectious individual for 120 min is 8.8 to 20.7%.

Discussion

The scarcity of scientific evidence available to underpin ventilation guidelines in hospitals makes modelling studies an attractive means of developing customised airborne infection control policies. In our study, we have focussed on producing conservative risk estimates that reflect the real-world activities of individuals (staff, patients and visitors) at the study location. It is important to consider that our risk estimates are expressed as percentages, and whilst this is a convenient and intuitive metric, it may not translate to a significant absolute number of infections in a room with low occupancy.[19,23]

Examining the points at which the existing air exchange rate in the Lung Function Laboratory intersects the curves shown in Figures 1a and b, it is clear that increasing air exchange further would provide a negligible reduction in an already very low infection risk. The attendant

increase in energy consumption required would be difficult to justify when the size of the room and its typical occupancy of up to 10 persons are considered.[24] A similar situation exists in the ED Isolation Room, where occupancy is low and outdoor air exchange is very high; even in the 'worst-case' scenario, a very low risk of 0.3% was estimated. The ventilation rate in the ED isolation room afforded substantial protection from the three pathogens modelled, and was approximately twice what is recommended by the Centers for Disease Control and Prevention for airborne infection isolation rooms.[25] Although the real-world bases of prescribed ventilation guidelines are limited, in these two scenarios modelling demonstrated that measured air exchange limited infection risk to relatively low levels. For both clinical rooms, in the case of a highly contagious airborne infection and/or highly susceptible group, modelling could be useful when conducted on a case-by-case basis to assess the benefit of increasing air exchange on infection risk.

Infections arising from airborne transmission during time spent in a physician's waiting or consulting room after the departure of an infectious individual have been documented previously.[3,4] However, these instances resulted from a combination of a paediatric source case and highly infectious airborne pathogen (measles), which were compounded by low outdoor air ventilation rates. Airborne transmission is widely acknowledged as the mechanism of spread of tuberculosis, although its role in influenza and rhinovirus transmission is less well-established.[16] There is, however, evidence to support increased airborne transmission of the latter two diseases under conditions where outdoor air exchange is low.[7,15] Our results suggest that the risk of influenza infection for susceptible individuals entering an Outpatient Consulting Room, whilst relatively low, are not negligible despite the total and outdoor air change rates meeting guidelines recommended for patient rooms and general wards.[10] This further emphasises the need to develop a rigorous scientific basis for

prescribing minimum ventilation rates within a diverse range of hospital environments.[9] It is also worthwhile to note that natural ventilation can reduce airborne infection risk and energy consumption compared to mechanical ventilation,[26,27] although this requires a climate amenable to this practice and appropriate planning of the hospital environment.

While ventilation rates increased in Outpatient Consulting Rooms A and B when doors were opened compared to closed, the increase was approximately 50% greater in Room A. Although not modelled, the risks to a susceptible individual entering Room B are therefore greater than those shown in Figure 2b, and such room-specific idiosyncrasies highlight the potential pitfalls of generalising results, even between two proximate and similar rooms. Notwithstanding this, infection risks in both rooms could be further reduced by allowing their doors to remain open for greater periods following occupation by a potentially infectious individual. However, reducing risk in one room occupied by a handful of individuals at the expense of increased risk in more densely occupied adjacent areas (e.g. waiting room) would represent a clearly false economy. The infectiousness of the pathogen, air volume into which it would be mixed and number of susceptible persons located nearby would need to be carefully considered and balanced against existing risks before opening doors could be recommended as a general control strategy. Further research addressing this issue is required.

Certain locations within hospitals are likely to be airborne infection 'hotspots', especially those where large numbers of untriaged individuals assemble. Beggs et al.[23] estimated risks of airborne transmission of TB, influenza and measles in a hypothetical hospital waiting area containing a single infectious individual. They found respective mean risks of infection for susceptible persons of 0.3, 2.6 and 13.5% for a 30 minute wait, and 0.8 and 6.6 and 30.9% for

a 60 minute wait. Despite differences in methodology, our work and that of Beggs et al.[23] highlight the approximate relative risks posed to individuals during the time spent waiting for and during medical consultations. The risk of an individual acquiring influenza appears more likely to occur during the actual consultation than the period spent in a waiting room based on the limited scenarios modelled in the two studies.

Infection risk modelling using the W-R or G-N models has several limitations that reflect the varying degrees to which its assumptions represent real-world conditions, and these are discussed in detail elsewhere.[19,20,23,24] The primary limitation is the reliance on quanta generation rates that have been calculated by a handful of prior epidemiologic investigations, although recent work suggests that this can be ameliorated somewhat by adopting a stochastic approach.[23] We have sought to minimise this limitation by using values approximately representative of median cases reported by the literature.[5,21] Nonetheless, it is prudent to view the output of infection risk models from a relative perspective.[23-24,26] Also, we did not assess risks posed to health care workers[6,28] which are undoubtedly higher than those presented here given their longer exposure times.

In locations with suitably accurate ventilation control systems, it may be possible to infer outdoor air exchange for some rooms from known total air flow rates and outdoor air intake percentages, thus enabling real-time infection risk estimates to be calculated when combined with occupancy at a given time. However, such an approach would need to be capable of representing air exchange rates at the room, rather than AHU, scale, and would only be appropriate for locations where air exchange due to non-mechanical means (e.g. infiltration) is small relative to that delivered mechanically. For greatest accuracy, a measurement-

oriented approach should be employed, even if only to validate the utility of the above method.

This study has built upon previous work by estimating airborne infection risk posed to individuals both simultaneous and subsequent to the presence of an infectious person. Infection risk was found to vary considerably in the different locations assessed. A simple model provided useful information regarding relative infection risks, and the role of room ventilation as a determinant of these. The influenza infection risk of susceptible individuals entering an outpatient consultation room after the departure of an infectious person was related to the occupancy time of both parties and the outdoor air exchange rate. Allowing the door to remain open for longer periods between consultations in the room we investigated could reduce transmission risk by significantly increasing air exchange rate. However, such a basic infection control strategy cannot be recommended without an appropriately detailed assessment of its effects on infection risks in proximate areas.

We have highlighted the utility of a customised approach that accounts for typical occupancy patterns of individuals at our study site. Ventilation measurements and modelling can be used to produce location-specific risk estimates that err on the side of caution and inform airborne infection control and patient management practices. Such an approach may increasingly find applications in the wake of the 2009 H1N1 pandemic, and in locations dealing with particularly susceptible individuals.

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Figure captions (Figure 1a to be stacked on top of figure 1b. Same for 2a and 2b)

Figure 1. Part (a) shows modelled influenza, TB and rhinovirus infection risk as a function of outdoor air exchange rate for individuals occupying the Lung Function Laboratory for 15 minutes in the presence of an infectious person. The current outdoor air exchange rate is indicated by the vertical dotted line. Part (b) shows equivalent risks for 45 minutes of exposure.

Figure 2. Part (a) shows modelled influenza quanta concentration in Outpatient Consulting Room A for consultations with an infectious individual for up to 120 minutes. The decay in quanta concentration following the departure of the infectious person after 15, 60 and 120 minute consultations , including an initial 5 minute period with the door open, is shown. Part (b) shows the corresponding infection risks for a susceptible person entering the room after the 5 minute open door period and remaining there for up to 120 minutes.

Appendix

Equations

In addition to equations (1) and (2) described in the text, we also used the following equations (see main text for nomenclature):

Total number of quanta within a room under steady-state conditions, n :

$$n = \frac{qI}{Q} \times V \quad (3)$$

The quanta concentration (q/m^3) in the room at time *t* (n_t) whilst occupied by an infectious person:

$$n_t = \frac{\frac{qI}{N} + \left(n_0 - \frac{qI}{N}\right)e^{-Nt}}{V} \quad (4)$$

The quanta concentration (q/m^3) in the room at time t_2 (n_{t_2}) following the departure of an infectious person at time t_1[4]:

$$n_{t_2} = n_{t_1} \times e^{-N(t_2 - t_1)} \quad (5)$$

Modelling approach

To calculate risk estimates that were conservative (i.e. not likely to be underestimates), we used the steady-state variation of the G-N model by setting n_0 to the steady-state value

(equation 3) for situations where the initial quanta concentration was lower than its steady-state value.[19] Under these conditions, G-N and W-R model outputs are the same. When modelling risk to an individual entering a room after the departure of infectious individual, steady-state, initial and decaying quanta concentrations were calculated using equations 3, 4 and 5, and the G-N model shown as equation 2 was used.