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Modelling the transmission of airborne infections in enclosed spaces

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SUMMARY

The Wells–Riley equation for modelling airborne infection in indoor environments is incorporated into an SEIR epidemic model with a short incubation period to simulate the transmission dynamics of airborne infectious diseases in ventilated rooms. The model enables the effect of environmental factors such as the ventilation rate and the room occupancy to be examined, and allows the long-term impact of infection control measures to be assessed. A theoretical parametric study is carried out to demonstrate how changes to both the physical environment and infection control procedures may potentially limit the spread of short-incubation-period airborne infections in indoor environments such as hospitals.

INTRODUCTION

The indoor air in buildings can play a significant role in the transmission of a wide range of infections. Aerosolized infectious agents may be introduced to the air by room occupants through actions such as coughing, sneezing and, in some instances – such as norovirus infection – vomiting. These microorganism-bearing droplets evaporate rapidly to form droplet nuclei, which with a typical diameter of $< 5\,\mu$m, can remain suspended in air for many hours. Ventilation systems and convection currents within rooms can disperse droplet nuclei over a wide area, with the potential to infect other occupants.

Infections that are known to be transmitted primarily by an airborne route include viral diseases such as measles and influenza and bacterial infections such as tuberculosis. In some emerging infections, for example those caused by the human metapneumovirus and the SARS-associated coronavirus the airborne route of transmission is also thought to be important. In addition to these communicable diseases there is increasing evidence that airborne transmission may play a role in the dissemination of many opportunistic pathogens responsible for a range nosocomially acquired infections. In particular airborne transmission has been implicated in nosocomial outbreaks of *Staphylococcus aureus*, including methicillin-resistant strains (MRSA) [1–3], *Acinetobacter* spp. [4, 5] and *Serratia marcescens* [6]. It is also thought that the high attack rates during norovirus outbreaks may be due to dispersion via aerosols [7]. It has been estimated that 10–20% of all nosocomial infections are spread by this route [8] which equates to a cost to the National Health Service in England in excess of £100 million annually [9].

These concerns together with the more recent threat of bioterroism attacks involving the deliberate release

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of an airborne infectious agent [10, 11] have prompted a resurgence of interest in measures to control airborne pathogens. Methods that have recently been discussed by researchers in the area include improving mechanical ventilation [12], use of personal protective equipment [13] and the use of ultraviolet germicidal irradiation (UVGI) devices both within rooms [14, 15] and in air-conditioning ducts [16]. For example experimental investigations have shown that the introduction of UVGI lamps reduces the levels of infectious material present in the air [17, 18]. Field trials [14, 16] have also yielded results suggesting that UVGI systems may have a beneficial impact.

Despite this interest, little work has been done to evaluate the overall benefits, particularly in developing epidemiological models that could be used to assess the long-term impacts of introducing new interventions. Models for examining infection in confined spaces are generally based on the work of Wells [19] and Riley *et al*. [20], and are limited to describing only the number of new infections for a fixed number of infectors not the full dynamics of an epidemic. Riley *et al*. did apply their model to a measles outbreak, but used discrete time steps based on the incubation period to approximate subsequent generations of infectors. We have presented a first step to addressing this limitation by combining ventilation-based models of indoor airborne transmission [13, 20] with classical SIR epidemic models [21, 22] that include an incubation period. The resulting systems of equations are used in a theoretical study to model the dynamics of an airborne infection in an environment such as a hospital. Through this parametric study it is demonstrated how a range of infection control measures may influence airborne disease transmission in enclosed environments.

SIR MODELS FOR A SHORT INCUBATION PERIOD DISEASE IN A VENTILATED SPACE

Epidemiological models for general disease transmission in populations have been used for many years and are well documented [21, 23]. The most common deterministic models are known as SIS and SIR models, and consist of systems of first-order differential equations describing the progression from susceptible (S) to infectious (I) individual. In an SIS model it is assumed that on recovery the individual becomes a susceptible again, whereas the SIR model considers that infectious people are removed from the transmission process either by death or isolation, or recovery to an immune state. The transfer between states in both models is governed by rate constants that are estimated from epidemiological data. These basic models have been used by numerous researchers and extended to include factors such as incubation periods, vaccination and immunity and interaction between different populations [21, 23]. They have also been applied to the spread of many diseases including tuberculosis [24] and the impact of HIV/AIDS on tuberculosis [25].

Many infectious diseases such as those caused by norovirus and influenza viruses that are problematic in institutions such as hospitals and nursing homes occur as distinct outbreaks over a relatively short timescale. For example the incubation period for influenza is typically 1–3 days and patients may then be infectious for a period of 4–6 days [26]. Norovirus, a major cause of gastroenteritis in hospitals throughout the world [7] that regularly leads to ward closures and staffing shortages [27] has a similar incubation period (1–2 days), however, the patient is usually only highly infectious for around 2 days [28]. The short incubation and infectious periods of these diseases are considered to be small enough that the population dynamics (admission and discharge rates) do not have to be included in a model. It is also assumed that people who become infected with, say, influenza, are not susceptible to re-infection on recovery (within the timescale of the model) and, therefore, an SIR type model is appropriate.

Basic SIR model

The classic deterministic SIR model, based on the work of Kermack & McKendrick [22], is given by three ordinary differential equations linking the change in number of susceptibles, S, the number of infectors, I, and the number removed through death, isolation or recovery to an immune state, R

$$\frac{\mathrm{dS}}{\mathrm{d}t} = -\beta \mathrm{SI}, \tag{1}$$

$$\frac{\mathrm{dI}}{\mathrm{d}t} = \beta \mathrm{SI} - \gamma \mathrm{I}, \tag{2}$$

$$\frac{\mathrm{dR}}{\mathrm{d}t} = \gamma \mathrm{I}, \tag{3}$$

$$\mathrm{S} + \mathrm{I} + \mathrm{R} = N, \tag{4}$$

Here N is the total population size, β is the contact rate between susceptibles and infectors, and γ is the

removal or recovery rate. By non-dimensionalizing the variables as

$$u=\frac{\mathrm{S}}{N},\quad v=\frac{\mathrm{I}}{N},\quad w=\frac{\mathrm{R}}{N},\quad \tau=\gamma t,\quad R_0=\frac{\beta N}{\gamma}, \qquad (5)$$

the model can be re-written as

$$\frac{\mathrm{d}u}{\mathrm{d}\tau}=-R_0uv,\quad \frac{\mathrm{d}v}{\mathrm{d}\tau}=(R_0u-1)v,\quad \frac{\mathrm{d}w}{\mathrm{d}\tau}=v, \qquad (6)$$

R_0 is known as the basic reproductive ratio and describes the average number of new cases that an infector produces in a particular population. When $R_0<1$ the disease dies out, while $R_0>1$ indicates that the infection rate is greater than the removal rate which may potentially lead to an epidemic.

Effect of the environment

Mathematical models examining airborne infection in confined spaces were first considered by Wells [19]. He introduced a unit of infection termed a 'quantum', defined as the amount of infectious material to infect $1-(1/\mathrm{e})$ (i.e. 63·2%) of the people in an enclosed space. Despite its stochastic definition, the number of quanta in a room is generally considered to be a physical measure of the infectious material present, which effectively indicates both the quantity and pathogenicity of an infectious material present in the air as well as the average susceptibility of a susceptible person. Wells published equations based on the quanta unit which showed a dependence of the number of new cases on the size of the space as well as the number of infectors, I, and susceptibles, S. Riley *et al.* [20] modified this model, to give an expression known as the Wells–Riley equation, reflecting the exponential increase in the number of new cases, C, with time for steady-state quanta levels in a room space.

$$C=\mathrm{S}(1-\mathrm{e}^{-(\mathrm{I}pqt/AV)}). \qquad (7)$$

Here, A is the ventilation rate in air changes per hour (AC/h), V is the room volume (m³), p is the average pulmonary ventilation rate of the susceptibles (m³/h) and q is the quanta production rate per infector (quanta/h). A further modification was introduced by Gammaitoni & Nucci [29] who published expressions linking the rate of infection with the room ventilation rate for non-steady state cases. They then used these expressions [13] to assess risks in clinical procedures where the room was initially considered to be clean. A review of the above models by Beggs *et al.* [30] demonstrated their range of applicability and used Gammaitoni & Nucci's general equation to evaluate the effects of room size, occupancy and ventilation conditions on the number of new infections.

The effect of the indoor environment can be examined by considering Gammaitoni & Nucci's [13, 29] equations relating the rate of infections in a ventilated space with a volume V (m³), and a ventilation rate of A (AC/h).

$$\frac{\mathrm{dS}}{\mathrm{d}t}=\frac{-p}{V}Q\mathrm{S}, \qquad (8)$$

$$\frac{\mathrm{d}Q}{\mathrm{d}t}=-AQ+q\mathrm{I}, \qquad (9)$$

In this case Q is the total quanta level in the space. Assuming continuous generation of quanta by the infectors and steady-state ventilation ($\mathrm{d}Q/\mathrm{d}t=0$), the level of quanta in the space is given by

$$Q=\frac{q\mathrm{I}}{A} \qquad (10)$$

and equation (8) becomes

$$\frac{\mathrm{dS}}{\mathrm{d}t}=\frac{-pq}{VA}\mathrm{IS}. \qquad (11)$$

This rate of reduction of susceptibles in equation (11) as a result of new infections is in fact equivalent to the rate of increase in new cases given by the derivative of the Wells–Riley equation [equation (7)] with time.

Comparing equation (11) with equation (1) we see that the term (pq/VA) is equivalent to β in the basic SIR model. Therefore the two models can be combined to give the following expressions.

$$\frac{\mathrm{dS}}{\mathrm{d}t}=-\frac{pq}{VA}\mathrm{IS}, \qquad (12)$$

$$\frac{\mathrm{dI}}{\mathrm{d}t}=\frac{pq}{VA}\mathrm{IS}-\gamma\mathrm{I}, \qquad (13)$$

$$\frac{\mathrm{dR}}{\mathrm{d}t}=\gamma\mathrm{I}. \qquad (14)$$

This model can also be represented by the dimensionless equations above but with the basic reproductive ratio now defined by

$$R_0=\frac{pq}{VA}\frac{N}{\gamma}. \qquad (15)$$

The model combines the effect of the physical environment with equations describing the progression of the disease and provides a means of quantifying the contact rate in terms of the room ventilation,

environmental conditions and level of airborne infectious material in the space. The definition of R_0 in these terms enables the effect of ventilation and room parameters on the disease transmission to be compared for different cases.

Inclusion of a short incubation period

Although the above model can give some useful indications about airborne disease transmission and the influence of physical parameters, it is difficult to apply to real situations as most diseases have an incubation period before the infected person becomes infectious to others. This limitation can be addressed by extending the SIR model for a ventilated room [equations (12)–(14)] to include an incubation period by assuming the susceptible is initially transferred to an exposed state, E, before going on to become an infector. The model is now referred to as an SEIR model, and the description becomes

$$\frac{\mathrm{dS}}{\mathrm{d}t} = -\frac{pq}{VA}\mathrm{IS}, \tag{16}$$

$$\frac{\mathrm{dE}}{\mathrm{d}t} = \frac{pq}{VA}\mathrm{IS} - \alpha\mathrm{E}, \tag{17}$$

$$\frac{\mathrm{dI}}{\mathrm{d}t} = \alpha\mathrm{E} - \gamma\mathrm{I}, \tag{18}$$

$$\frac{\mathrm{dR}}{\mathrm{d}t} = \gamma\mathrm{I}, \tag{19}$$

$$\mathrm{S} + \mathrm{I} + \mathrm{E} + \mathrm{R} = N, \tag{20}$$

where α is the progression rate from exposed to infector, equivalent to the reciprocal of the incubation period.

As previously this model can be re-written in terms of non-dimensional variables

$$\left.\begin{array}{lll} u = \dfrac{\mathrm{S}}{N}, & x = \dfrac{\mathrm{E}}{N}, & v = \dfrac{\mathrm{I}}{N}, \\ w = \dfrac{\mathrm{R}}{N}, & \tau = \gamma t, & \sigma = \dfrac{\alpha}{\gamma}, \end{array}\right\} \tag{21}$$

to give

$$\left.\begin{array}{ll} \dfrac{\mathrm{d}u}{\mathrm{d}\tau} = -R_0 uv, & \dfrac{\mathrm{d}x}{\mathrm{d}\tau} = R_0 uv - \sigma x, \\ \dfrac{\mathrm{d}v}{\mathrm{d}\tau} = \sigma x - v, & \dfrac{\mathrm{d}w}{\mathrm{d}\tau} = v. \end{array}\right\} \tag{22}$$

The reproductive ratio remains unchanged from equation (15) as the total number of new infections depends on the rate at which an infector produces new cases with respect to the removal rate, which is not affected by a delay in a susceptible person becoming infective.

BEHAVIOUR OF SHORT INCUBATION PERIOD SEIR MODEL

The SEIR model described above [equations (16)–(19)] can be used in a theoretical study to examine how changes in the ward environment may influence outbreaks of short-incubation-period diseases. As the models are systems of nonlinear differential equations, solutions are most easily found using numerical methods. In this study the mathematical analysis package Maple v.9 (MaplesoFt, Waterloo, Canada) is used to numerically solve the governing equations and produce typical epidemic curves showing the probable disease progression with time.

Choice of parameters

The study was based on the initial conditions and parameter ranges given in the Table.

The numbers of people are intended to be representative of an area of a hospital with, say, 100 patients on a number of connected wards and 100 health-care workers/other people. Each patient is assumed to occupy a volume of 36 m^3 ($3 \times 4 \times 3$ m), with the remainder of the area (corridors, offices, nurses' rooms, treatment rooms, etc.) occupying the same space again giving a total volume of 7200 m^3. A pulmonary ventilation rate of 8 l/min is typical for an adult [31]. The model assumes the population is constant during the outbreak and that the air in the wards is fully mixed. Although the population may not be constant in some situations, in others, such as norovirus outbreaks, wards are usually closed to new admissions and discharges are postponed, therefore, this assumption is reasonable under these circumstances. Likewise the assumption that the air is fully mixed may not be appropriate for all situations. However, both these assumptions enable a first approximation to be made about the transmission dynamics of airborne infection in an enclosed area, and further possible improvements are outlined in the discussion. The values for α and γ are based on data for the incubation time and periods of infectivity for influenza and norovirus as given above.

The value for the quanta production rate, q, in any disease outbreak is the parameter that is the most

Table. *Basic conditions and study parameter ranges for SIR model simulations*

Parameter	Base value	Study range
S (at $t=0$)	199	99–199
I (at $t=0$)	1	1
E (at $t=0$)	0	0
R (at $t=0$)	0	0
A	3 AC/h	3–8 AC/h
q	10 quanta/h	1–50 quanta/h
V	7200 m^3	7200 m^3
p	0·48 m^3/h (8 l/min)	0·06–0·48 m^3/h
α	1/day (1-day incubation period)	1–0·33/day (1- to 3-day incubation period)
γ	0·5/day (2-day infectious period)	0·5–0·166/day (2- to 6-day infectious period)

difficult to quantify. To date, most of the quanta values presented in the literature relate to tuberculosis [12, 13] and very little data have been published relating to quanta production rates for short-incubation-period infections that may be more of an issue in a typical hospital environment. Rudnick & Milton [31] estimated values of quanta production rate for rhinovirus as 1–10 quanta/h and influenza as 15–128 quanta/h depending on the calculation method. Riley *et al.* [20] calculated a value of 570 quanta/h for a typical measles case, which is consistent with the high transmission rates of this disease in school outbreaks. For the purposes of this study, quanta production values of 1–50 quanta/h are used as suitable values to examine the behaviour of the model.

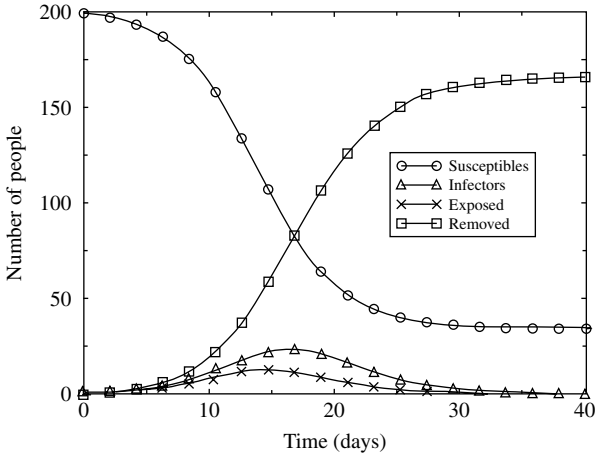

Fig. 1. Predicted dynamics of an outbreak of an airborne infection with the disease and environment characteristics given by the base parameters in the Table.

Dynamics of an outbreak

Figure 1 shows a typical result from the SEIR model for a theoretical airborne infection given by the base parameters in the Table. The epidemic curves produced are characteristic of those produced by all SEIR models. Initially the number of susceptibles falls with time, with the numbers of infectors, exposed and removals all increasing. The outbreak peaks after about 16 days, with around 25 active infectors, and the rate of change of both susceptibles and removals at a maximum. After this point the outbreak starts to wane, with all the variables levelling off to constant values. The outbreak is over after about 35 days, with approximately 20% of the susceptibles remaining uninfected. The curve indicating the exposed group is a similar shape to the infector profile over the period, but peaks 1 day (the incubation period) earlier at a lower value. R_0 for this particular case is 2·133, a value that is indicative of an epidemic.

Impact of physical environment

The model is first used to examine how changes to the physical environment, in particular the ventilation rate and the occupancy level, may affect the course of an outbreak of an airborne infection. Figures 2 and 3 show the effect of increasing the ventilation rate on the course of the outbreak modelled in Figure 1. In Figure 2 the ventilation rate is increased to 5 AC/h. This has the effect of increasing the duration of the outbreak, yet it reduces the total number of cases. The reproductive number is now $R_0 = 1{\cdot}28$, reflecting

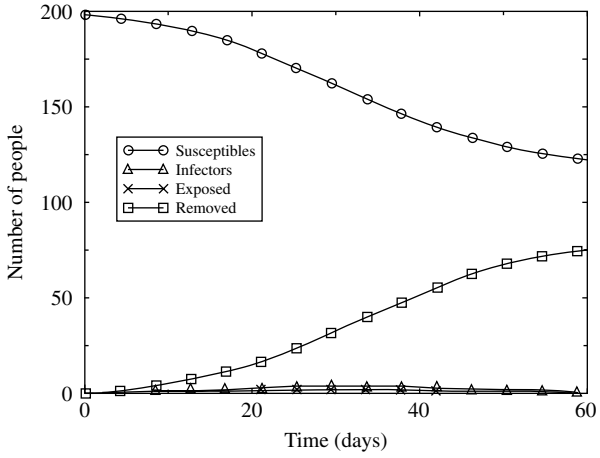

Fig. 2. Predicted progression of the epidemic modelled in Figure 1, with the ventilation rate increased to 5 AC/h.

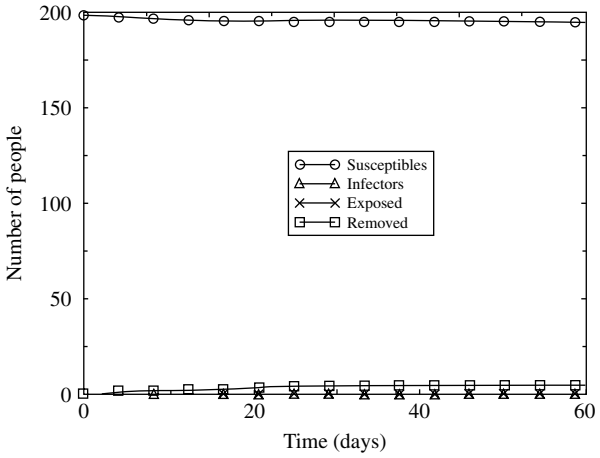

Fig. 3. Predicted progression of the epidemic modelled in Figure 1, with the ventilation rate increased to 8 AC/h.

the lower transmission rate for the disease. The rate of infection is much slower and the peak number of infectors significantly lower.

In Figure 3 the ventilation rate is 8 AC/h, which has a dramatic effect on the dynamics of the outbreak with less than 10% of the susceptibles infected. This phenomenon is always seen with SIR-type models when the contact rate is smaller than the removal rate ($R_0 = 0{\cdot}799$), effectively meaning the infectors recover before they have the chance to infect anyone else.

The impact of the ventilation rate can also be examined by considering the reproductive number, R_0. Figure 4 shows how R_0 changes with the ventilation rate, again for the base parameters in the Table, and also with an occupancy of 50%. It can be seen that with 200 occupants (S = 199, I = 1), a ventilation rate below 6·4 AC/h results in a value of $R_0 > 1$ and the potential for this infection to become an epidemic. With only 100 occupants (S = 99, I = 1), a ventilation rate of half this value will lead to the same conditions.

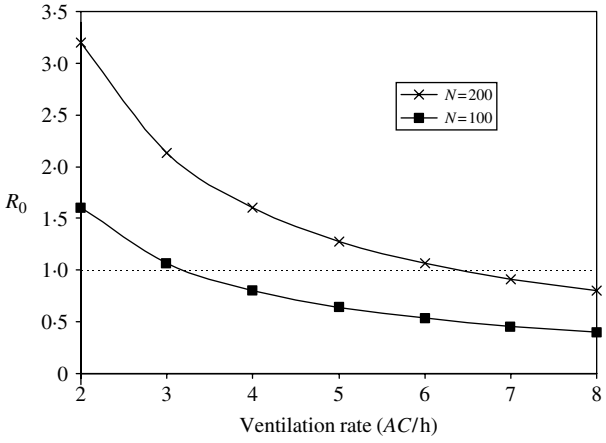

Fig. 4. Impact of ventilation and ward occupancy on the potential for an epidemic, for the base conditions in the Table.

Impact of disease and infector/susceptible characteristics

Further examination of the model can reveal how the characteristics of the infection and the infectors and susceptibles may affect the progression of an outbreak of an airborne infection. Increasing the incubation period of an infection has the result of increasing the duration of the outbreak, but it does not impact on the overall number of people infected, and the reproductive number remains unchanged. However, this may have implications for infection control procedures as outlined in the Discussion section.

The influence of both the disease itself and the infector and susceptible characteristics can be examined by considering the recovery rate, γ, the pulmonary ventilation rate, p, and the quanta production rate, q, which incorporates the infectivity of the pathogen, the response of the average susceptible and the ability of the average infector to disseminate the infection into the room in an airborne state. It can be seen from equation (15) that R_0 is directly proportional to both the infectious period ($1/\gamma$) and the quanta production rate. Hence, as expected, infections characterized by a low quanta production rate require a much longer infectious period for an outbreak to reach epidemic levels ($R_0 > 1$) than those infections with a high quanta production rate.

The relationship between R_0 and quanta production rate for three pulmonary ventilation rates

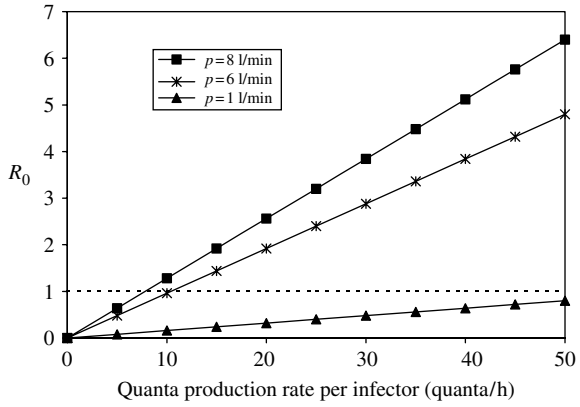

Fig. 5. Effect of quanta production rate on reproductive number at different pulmonary ventilation rate.

is also linear as demonstrated by Figure 5. A value of $p=8$ l/min is typical of a normal adult, while $p=6$ l/min is intended to be representative of a child. In both of these cases the reproductive number is directly proportional to the quanta production rate and exceeds $R_0=1$ when $q=8$–10 quanta/h indicating the possibility of an epidemic. However, when the value of p is reduced to 1 l/min even at high quanta production rates the reproductive number does not exceed $R_0=1$.

DISCUSSION

The models and results outlined in this study have been used to demonstrate the probable dynamics of airborne infections and the impacts of changes in the physical environment or disease characteristics. However, these results can also be interpreted in the context of infection control measures. The results presented in Figures 2 and 3 suggest that increasing the ventilation rate may reduce the rate of infection and that high ventilation rates may remove the potential for an epidemic altogether. In a real situation it is likely that a moderate increase in ventilation rate may make an outbreak more manageable, with for example fewer problems with staffing shortages and a higher possibility of isolating the smaller numbers of infectors. Reducing the ward occupancy density was also shown to have a similar impact on infection rates, with the results in Figure 4 indicting that much lower ventilation rates were necessary to prevent an outbreak with only half the original number of occupants.

The effect of both the quanta production rate and the infectious period of the disease, also have implications for infection control procedures in the event of an outbreak. For infections that have relatively low quanta production rates, isolation of infectors may be an effective means of preventing an epidemic, particularly where the disease has a long infectious period. For example, using the base parameters in the Table where an infectious patient is emitting 10 quanta/h and the infectious period of the disease is 2 days, $R_0=2{\cdot}133$, suggesting an epidemic is likely to occur. If the patients are diagnosed and isolated within 1 day, the effective reduction in the infectious period results in $R_0<1$ and may prevent an epidemic. However, for highly contagious diseases, indicated by high quanta production rates, epidemic conditions may be present with an infectious period of less than 8 h. In these cases it may be impossible to isolate individual cases quickly enough and it may be necessary to isolate whole wards or units as seen in norovirus outbreaks [27].

The effects of incubation and infectious period described here are not unique to this model for airborne transmission, and are seen with all SIR models that incorporate an incubation period. However, they are still important to consider in the context of airborne infections, as it is likely that the transmission rate, $\beta=pq/VA$, will be different than for many infections transmitted via contact routes. The rate may also be less controllable as isolating patients with airborne infections is more involved than for those with infections that are only transmitted by contact, generally requiring the use of a negatively pressurized isolation room [32].

The final aspect of the parametric study is also relevant to infection control procedures. Although the pulmonary ventilation rate 1 l/min plotted in Figure 5 is an unrealistic breathing rate for a normal person, it may be equivalent to introducing some form of protection such as facemasks. In this case p can be considered to be the rate at which a person breathes only the contaminated air. Gammaitoni & Nucci [13] considered this intervention in their modelling and suggested that surgical and HEPA masks reduce the pulmonary ventilation of contaminated air to the equivalent of $0{\cdot}6p$ and $0{\cdot}03p$ respectively. A value of $p=1$ l/min in Figure 5 is $0{\cdot}125$ of the original value of 8 l/min for an adult, and is therefore representative of the level of reduction that the use of masks may achieve.

Although the results presented in this study are all for theoretical cases, they demonstrate how infection control interventions may reduce the number of

infections, and possibly prevent an outbreak. The study has shown that the models can be used to examine a wide range of possible infection control measures, some of which require physical modifications such as changes to the ventilation system and others that are procedural such as isolation of patients or the use of face masks. This allows different controls to be compared and the most appropriate measures for the situation to be selected. The models may also be applied to evaluating further engineering infection control measures such as the application of upper room UVGI devices. The effectiveness of UVGI systems can be quantified in terms of an effective air change rate. For example Riley *et al.* [33] showed that the effectiveness of a 17 W UV fitting in their ventilated test room against airborne bacille Calmette–Guerin (BCG) was equivalent to an increase in the ventilation rate of 10 air changes per hour. The ability to make this comparison may be of benefit in situations where it is believed that increasing the ventilation rate will be beneficial, however, it is impractical or too expensive to fit a new air-conditioning system. In such cases the fitting of UV lamps could be a possible solution as they are easily installed in most buildings.

The model presented here does have a number of limitations. The equations are based on the assumption that the room air is fully mixed and, therefore, has a uniform distribution of quanta throughout the space. In reality most rooms are not well mixed and room air simulation results suggest that considerably higher infection concentrations will occur close to the source [30]. The risk of disease transmission is therefore likely to be greater for susceptibles in close proximity to the infectors. This was demonstrated by an outbreak of tuberculosis in an Arkansas hospital caused by aerosols generated by irrigation of a tuberculous abscess [34], where the prevalence of tuberculin reactivity decreased considerably with the distance from the source. Although increasing the ventilation rate in a space will in general reduce the bioburden in the air and hence the risk of infection for occupants, the design of ventilation systems may have a significant bearing on the actual distribution of bioaerosols in the space. For example previous computational and analytical studies [15, 35] have shown that the disinfection potential of devices such as UVGI is strongly related to the layout of the ventilation system, and a change in ventilation system design could have a negative impact on infection control.

The applicability of the model presented here is also limited by the assumption of a closed population and the deterministic nature of the equations. Although some populations may be assumed to have no inputs and outputs, particularly over short periods of time, the assumption is not strictly valid for many real hospital wards, especially where there are significant numbers of visitors and a high patient throughput. In these cases it is easily possible to extend the model to include admission and discharge rates, such as those used to simulate the transmission of tuberculosis [24]. For more complex situations it would also be possible to model the staff, patients and visitors as separate patient groups and include the interaction between them such as in Cooper *et al.* [36]. The issue of the deterministic nature of the model is significant in situations where the risk of infection may be influenced by chance events as much as by the environment, such as when the number of individuals involved in an outbreak is small. The deterministic model presented here will still give useful indicators in these cases, particularly when comparing infection control measures. However, it is possible to use similar assumptions to formulate stochastic SIR models [21, 23], which may be more appropriate for some cases.

When using this model, parameters such as the room size, room ventilation rate and pulmonary ventilation rate can all be calculated or estimated for a particular case with a reasonable level of confidence. However, determining a suitable value for the quanta is much more difficult. The fact that the concept of the quanta encompasses the infectivity and virulence of a given strain of a pathogenic microorganism, as well the susceptible and infector characteristics, means that even with the same infection there are likely to be wide variations in suitable values. It is also likely that in reality an infector will not remain at the same level of infectiousness throughout their illness, but will become less infectious as they start to recover. Most of the values quoted in the literature relate to tuberculosis outbreaks [12, 13] and are calculated by applying models such as equations (8) and (9) to actual cases to find an average value for the quanta. Values collated by Beggs *et al.* [30] for several tuberculosis outbreaks indicated the large range of quanta values that may be associated with a single disease. For example typical tuberculosis patients were seen to generate quanta levels of the order of 1·25 quanta/h, however, some cases were noticeably more infective, with quanta levels of up to

60 quanta/h. However, these values were all much lower than the values calculated following outbreaks associated with a range of clinical procedures. Quanta values calculated by Gammaitoni & Nucci [13] included 360 quanta/h for a bronchoscopy-related outbreak [37] and 2280 quanta/h following a hip abscess irrigation [34], suggesting that some clinical procedures may create significant numbers of aerosolized microorganisms. For predicting the likelihood of epidemics for other airborne infections, where a suitable quanta value is not known, a similar calculation method to that used by Nardell *et al*. [12] can be used by applying the model to a previous outbreak and selecting a value of quanta such that the results approximate to the progression of the infection seen in reality. Alternatively predictions can be made at a range of quanta values to evaluate the impact of interventions for a range of cases.

Despite the limitations of the model, the predicted results suggest that changes in the physical environment may lead to a long-term reduction in infections and potentially prevent epidemics. This is in agreement with findings from several studies examining the impact of interventions such as UVGI lamps. For example Wells *et al*. [38] investigating the impact of UV air disinfection in schools over a 5-year period showed a consistently lower incidence of measles in the irradiated schools. Evidence is also given by Menzies *et al*. [16], who showed in a double-blind study that the incidence of a range of symptoms, including respiratory complaints, decreased following the introduction of UV air disinfection in the air-conditioning ducts of an office building. Hence the model described here will, therefore, at the very least give an indication of the impact of various factors in the physical environment.

CONCLUSIONS

The model developed in this study shows how environmental factors may be included in classical epidemic models to examine the impact of changes in the physical environment and disease characteristics on the transmission of airborne infection in ventilated rooms. The parametric study has shown that the model can be used to examine a range of infection control measures and the results suggest that the most suitable method depends on both the infection characteristics and the physical environment.

Although the model developed here is relatively simple, the same methodology could easily be applied to the more complex epidemic models in the literature, including stochastic models suitable for small populations [21] and the models for tuberculosis spread with factors such as HIV/AIDS [25].

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DECLARATION OF INTEREST

None.

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