Impact of environmental conditions on airborne disease progression

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Abstract

Small droplet nuclei can stay in air over hours without disturbance like ventilation. Pathogen loaded particles can easily build up in closed spaces after being produced by infected people, especially in poorly ventilated conditions. Thus, superspreading events can happen in indoor environments, such as the ones observed during the ongoing SARS-COV-2 pandemic. The likelihood of infection for airborne-diseases is different between indoor and outdoor environments. In this paper, we developed an SIR model to understand the dynamics in transmission for an airborne disease by combining both indoor and outdoor transmission. We evaluated how the proportion of individuals' groups changed as a function of movement frequency, indoor environmental conditions, and time. The results obtained highlighted the importance and impact of indoor conditions throughout the transmission of airborne diseases.

Introduction

Airborne transmission can be defined as the spread of an infectious agent as result of the dissemination of small droplet nuclei (defined as aerosols) which are normally produced during any expiratory activity. An important feature related to these aerosols is that they can remain suspended in air over long distances and periods due to their small particle size. Given their small particle size (under 100 μM) and due to their capacity to remain in the air, their concentration can build up in poorly ventilated spaces favoring the dissemination of an infectious agent(Jones & Brosseau, 2015).

Several respiratory infectious agents rely on airborne-transmission such as tuberculosis, influenza, measles, chicken pox and severe acute respiratory syndrome coronavirus 2 (SARS- CoV-2) (Xiaolei Gao et al., 2009). For example, during the early SARS-CoV-2 pandemic it was suggested that maintaining six feet could help to prevent the spread of COVID-19. The logic behind this idea relies on the fact that larger respiratory droplets (>100 μM) loaded with the pathogen are likely to be pulled down due to gravity within 1 or 2 m. However, recent studies have shown that SARS-CoV-2 can remain viable in small aerosols for several hours(Ho, 2021)and it has been detected in air samples from rooms and cars occupied by COVID-19 patients(Greenhalgh et al., 2021) .

The aforementioned helps to conclude that the likelihood for airborne-diseases transmission will be different between indoor and outdoor environments. More importantly several environmental factors such as temperature, relative humidity, airflow, ventilation, and air filtration of indoor environments can play an important role in superspreading events (Wang Chia C. et al., n.d.). A superspreading event occurs when a large number of secondary infections take place from a single infected individual (Althouse et al., 2020). Since they occur in closed environments where individuals spend most of their time, understanding the dynamics in transmission for an airborne disease should consider indoor environmental conditions in parameters estimation.

Each environmental factor affects the transmission in different ways. Temperature is relevant given that it can affect the stability of the cellular membrane components such as lipids and proteins. For example, epidemiological evidence suggests that transmission of upper airways viruses is favored at lower temperature (Wang Chia C. et al., n.d.).

Relative humidity modulates the evaporation rate and particle size of droplets and aerosols. There is an inversely proportional relationship between relative humidity and evaporation, the lower the relative

humidity the faster the evaporation. Nevertheless, the fitness of pathogens varies, and in some cases certain RNA viruses are capable of being stable at low relative humidity (Marr et al., 2019). Therefore, the viability of a pathogen and its relationship with the relative humidity is pathogen specific rather than general. Relative humidity is also a significant factor to consider when seasonality is meant to be explained, given that pathogens that are capable of being stable at very low relative humidity are known to cause outbreaks during winter periods (Božič & Kanduč, 2021).

The movement of large droplets, as mentioned previously, depends mainly on gravity, on the other hand aerosols are strongly influenced by airflow patterns, ventilation, and air filtration. The airflow in indoor environments relies on the design and functionality of natural and artificial ventilation systems (Parhizkar et al., 2021; Wang Chia C. et al., n.d.). Poorly ventilated spaces show high concentration of pollutants (such as pathogen-loaded aerosols). This elevated concentration comes from the low dilution capacity present in the closed space due the absence of an appropriate supply of outdoor air capable of displacing any pollution back to the outdoors. Thus, these factors should be considered when determining the risk of airborne infection, and subsequently as an intervention needed to mitigate the spread of airborne diseases.

Throughout this paper we presented a stratified SIR model, considering outdoor and indoor compartments. Furthermore, we developed a set of differential equations meant to explain the movement of individuals within compartments and the rate of infection for both groups. The results explained why we should pay attention to indoor transmission and how did ventilation condition affect indoor quanta concentration then influence the transmission dynamics.

Methods

SIR Model

The model used for the current study is displayed in **Figure 1.** The model is a stratified model where there are compartments for susceptible and infected individuals within an indoor and an outdoor environment. Given that indoor environments can count with low ventilation and limited space compared to outdoor environments, for an airborne disease, pathogen loaded particles may suspend in air for longer times, which increases people infection risk. Another feature of the model involves the movement within compartments of the same class of individuals. There is no consideration of different compartments for recovered individuals given that it is not relevant.

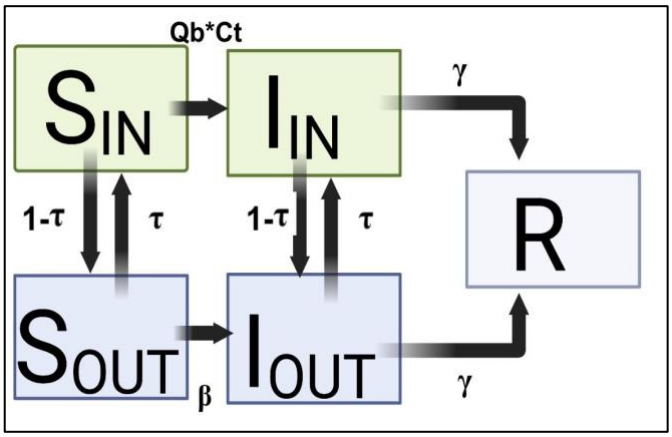

Figure 1: SIR stratified model

Mathematical equations for the model

The Wells-Riley equation (Eq.1) is useful for understanding the spread of airborne diseases considering indoor conditions. Where P is the probability of a new case of infection in a closed indoor environment and n accounts for pathogens inhaled by occupants indoors. In this way, n is proportional to the breathing rate (Qb) and the pathogen concentration (c_t) (Eq.2).Now, pathogen concentration can be understood as a function of time (Eq.3) that is dependent on the emission rate from the infector (E) which depends on the

number of Infected people, the volume of the indoor space (V) and the particle removal constant (ε). This removal constant (Eq.4.)(Parhizkar et al., 2021) is the one that is influenced by indoor conditions. Therefore, it is a function proportional to the air exchange rate normalized by space (λ), the deposition rate (Ks), and the decay of the pathogen as a function of temperature and humidity (Kd). Throughout this document for the parameter ε we will consider constant values

The probability of infection (P) can be considered as the proportion of new infection within an exposure period of time. Therefore, the product between Qb and Ct could be considered as an approximate estimate of the transmission rate (β) times Infected people in a conventional SIR model. Taking into account the previous, we could be able to develop a system of equations for an indoor SIR model (Eq.5) (Xiaolei Gao et al., 2009).

$$P = 1 - e^{-n} \quad \text{(Eq.1)}$$

$$n = Qb*t*\underline{C_t} \quad \text{(Eq.2)}$$

$$\frac{dCt}{dt} = \frac{E*I-\varepsilon Ct}{V} \quad \text{(Eq.3)}$$

$$\varepsilon = \lambda + Ks + Kd \quad \text{(Eq.4)}$$

$$\frac{dS}{dt} = \text{- Qb}C_t S \quad \text{(Eq.5.1)}$$

$$\frac{dI}{dt} = QbC_t S - \gamma I \quad \text{(Eq.5.2)}$$

$$\frac{dR}{dt} = \gamma I \quad \text{(Eq.5.3)}$$

As mentioned previously, individuals can move within compartments of the same class. This means that susceptible individuals can migrate from outdoors to indoor compartments and vice versa. This movement can be included into our model by considering constant fluctuations over time and the steady rise and fall of the cosine function makes it ideal for modeling them. We assumed that they followed function $f(\tau) = \boldsymbol{\tau}(\mathbf{1} + \boldsymbol{mcos}\left(\frac{\mathbf{2\pi\, t}}{\mathbf{24}}\right))$. In each hour, changes in the function depend on two constant parameters $\boldsymbol{\tau}$ and **m**. And the exchange rate repeated itself every 24 hours. The full system of equations is as follows:

$$\frac{dSi}{dt} = S_o\boldsymbol{\tau}(\mathbf{1} + \boldsymbol{mcos}(\frac{\mathbf{2\pi\, t}}{\mathbf{24}}) - QbC_tS_i - S_i(\mathbf{1} - \boldsymbol{\tau}(\mathbf{1} + \boldsymbol{mcos}(\frac{\mathbf{2\pi\, t}}{\mathbf{24}}))$$

$$\frac{dSo}{dt} = S_i(\mathbf{1} - \boldsymbol{\tau}(\mathbf{1} + \boldsymbol{mcos}(\frac{\mathbf{2\pi\, t}}{\mathbf{24}})) - \boldsymbol{\beta} S_o I_o - S_o\boldsymbol{\tau}(\mathbf{1} + \boldsymbol{mcos}(\frac{\mathbf{2\pi\, t}}{\mathbf{24}})$$

$$\frac{dIi}{dt} = QbC_tS_i + I_o\boldsymbol{\tau}(\mathbf{1} + \boldsymbol{mcos}(\frac{\mathbf{2\pi\, t}}{\mathbf{24}}) + QbC_tS_i - I_i(\mathbf{1} - \boldsymbol{\tau}(\mathbf{1} + \boldsymbol{mcos}(\frac{\mathbf{2\pi\, t}}{\mathbf{24}})) - I_i\gamma$$

$$\frac{dIo}{dt} = \boldsymbol{\beta} S_o I_o + I_i(\mathbf{1} - \boldsymbol{\tau}(\mathbf{1} + \boldsymbol{mcos}(\frac{\mathbf{2\pi\, t}}{\mathbf{24}})) - I_o\boldsymbol{\tau}(\mathbf{1} + \boldsymbol{mcos}(\frac{\mathbf{2\pi\, t}}{\mathbf{24}}) - I_o\gamma$$

$$\frac{dR}{dt} = \gamma(I_o + I_i)$$

Where subindexes i and o indicate indoor and outdoor respectively.

In the following table, in a time scale of an hour the parameters input used for the SIR model are summarized.

Parameter	Value	Parameter	Value
Average contact number **β (person/h)**	1/8	Breathing rate **Qb (m^3/h)**	0.48
Infectious period **1/γ (h)**	1/24	Quanta generation rate **E (q/h)**	48
Exchange frequency **τ (h)**	(0.2 - 0.6)	Room volume **V (m^3)**	180
Exchange frequency **m (h)**	0.5	Removal constant **ε (m^3/h)**	(0-50)

Table 1. Input parameters used

Based on the SIR model, for outdoor environment, the parameters considered for the progression of the disease were the traditional ones. In it the susceptible compartment was decreased at a rate of **β** and recovered compartment was increased at a rate of **γ**, where **β** is the transmission rate and **γ** is the recovery rate.

However, for indoor conditions, more assumptions were proposed. First, when infected people entered a closed space, the pathogen particles would be emitted at a rate **E**. Indoor ventilation, particle deposition and biological decay work together to decrease the particle concentration. Assuming that indoor spaces are well-mixed, the particle distribution was homogenous, and it happened in seconds. Therefore, exposure time between susceptible and infected individuals have a significant impact in infection dissemination. Furthermore, all individuals had equal possibility to contact pathogen particles because particles were evenly distributed. We also assumed that every quantum can certainly lead to infection once inhaled. So, transmission rate which originally depends on contact rate between infected and susceptible individuals, can be transformed to quanta inhaling rate, which is understood as breathing rate (Qb) times pathogen concentration (C). We also assumed closed population and long lasting immunity after infection.

Results and Discussion:

The duration of the modeling was 10 days. A 2*2 factorial design was carried out. One factor was the fluctuation in time within compartments of the same class (**τ**) assessed at three levels (0.2 - 0.4 - 0.6 (h)). The other factor was the removal constant (**ε**) assessed at six levels (0 - 5 - 10 - 15 - 25 - 50 (m^3/h)); these levels were assigned considering the air change rates values recommended for different indoor spaces by the United States environmental protection agency (EPA).

As shown in **Figure 2** the total indoor quanta concentration of expelled pathogen particles was determined for the different conditions. Variation was observed both within and between scenarios when removal rate varied. So, change in quanta concentration was as a function of **τ and ε**.

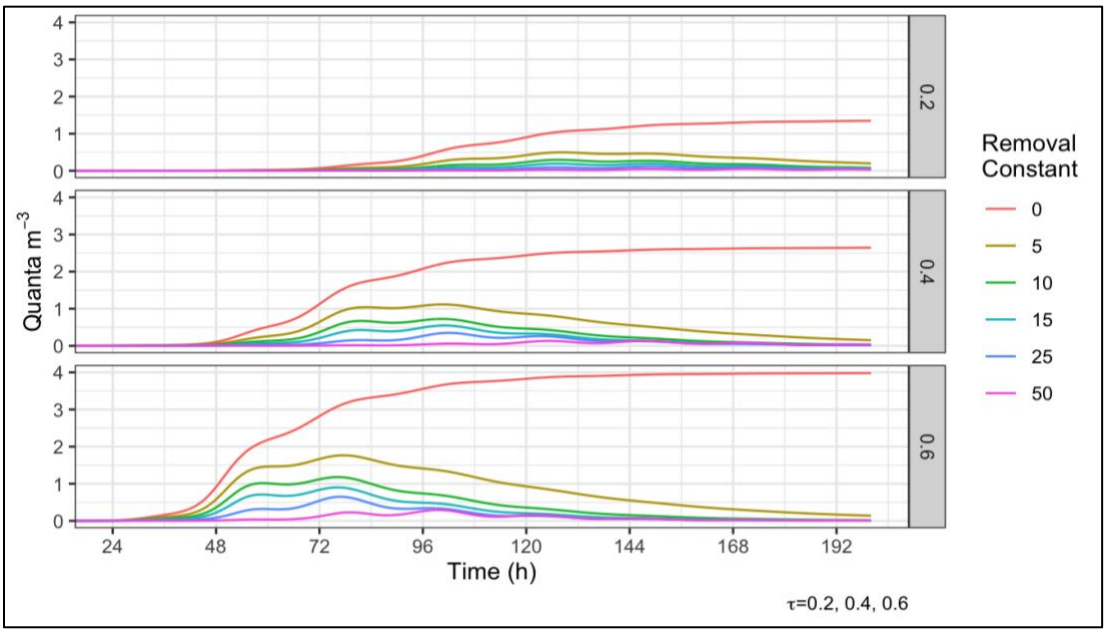

Figure 2: Quanta concentration under different conditions over time. τ = Exchange frequency between compartments. ε = Removal constant.

When the removal rate is 0, at **τ** = 0.6, the highest quanta concentration reached 4 quanta/m^3 at day 6 while when **τ** = 0.2, the highest quanta concentration was just around 1.5 quanta/m^3 at day 10. High population exchange rate was beneficial for having more particles suspended in air and reaching the highest quanta concentration . At same **τ** for the different levels of **ε**, quanta concentration decreased when removal rate increased. For lower removal rate, at same day, the quanta concentration was always higher, which meant lower removal rate was beneficial for keeping quanta suspended for longer time. For example, when **τ** = 0.6, quanta concentrations were back to zero at around day 6 for scenarios where removal rate = 10,15,25,50 per day. Even though at day 10, quanta concentration was still higher than zero for removal rate=5; on the other hand, for removal rate = 0, there was no evidence of any decline in quanta concentration. These findings highlight two main things, the impact of indoor conditions in the concentration of quanta in a closed space which directly correlates with an increased probability of infection based on the Wells-Riley equation. Also, it helps to understand how population exchange between spaces contributes to the buildup of pathogen loaded particles in indoor environments.

It is also observed that changes in the proportion of infected people were as well a function of **τ and ε**. At the same **τ** for different **ε**, it was shown in Figure 3 that low removal rate reached higher proportion of infected individuals in shorter period. For example, when the fluctuation rate equaled to 0.4 and the removal rate ranged from 0 to 50, the date when the infected proportion reached the highest point ranged from 3 to 6 days, respectively. At day 10, when infected proportion for other values of **ε** have reached zero, it was still above zero for removal rate equals to 50. Now proportion of infected people changed depending on the fluctuation rate. When **ε = 0,** at different **τ** the proportion of infected people and the time for peak of infection increased as the fluctuation rate increased. By looking at the red curve (**ε = 0**) when **τ** was 0.2 and 0.6 separately, it was found that peak occurred in day 5 and day 3, and the highest infected proportions were around 0.4 and 0.6, respectively. The abovementioned helps us to infer different things. The peak of infection for a whole population, changes as a function of the fluctuation rate between compartments and more importantly as a function of indoor conditions. Thus, optimization of indoor

conditions can help to delay the progression of a disease by a time frame that can be useful for the implementation of sanitary interventions that can prevent the outbreak of a disease.

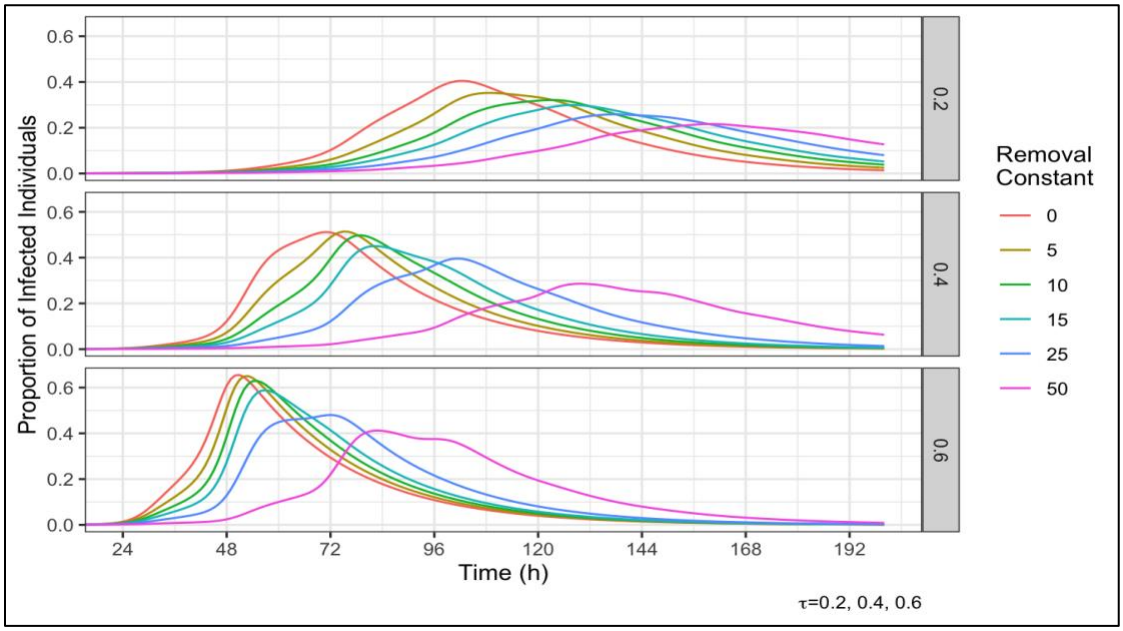

Figure 3: Proportion of infected individuals at different conditions over time. τ = Exchange frequency between compartments. ε = Removal constant.

From both figure 2 and 3, we could find that removal rate and population exchange rate played important roles in deciding disease transmission dynamics by affecting indoor particle concentrations. Besides outdoor disease transmission, which we mimicked by using traditional SIR model, attentions should also be paid to indoor disease transmission because people spent more time indoor in a daily base scenario regardless of occupation. Indoor disease transmission could highly affect when and how severe outbreaks will occur as observed in figure 3. As we hardly controlled people behavior, the population exchange rate could be regarded as a constant. To reduce indoor quanta concentration and decrease possible infected proportion, increase pathogen removal rate was necessary, namely we needed higher ventilation rate.

Limitations

We set the room spaces to $180m^3$. In reality $180m^3$ can at most contain 60 people if each of them occupied $1m^2$ on the ground and the height of the room is 3m. However, the model doesn't take population size into consideration, which means the infected proportion calculated can be based on an extremely large population size and incredible high indoor quanta concentration. In a word, the model overestimate infected proportion because any closed space has a maximum population containing capacity, which was not considered in our model.

The disease dynamics are highly dependent on initial conditions. In our model, we set the S_{in}=0.9998, I_{in}=0.0001, R=0, S_{out}=0.0, I_{out}=0.0001, C_0=0. However, we can't have all people in indoor conditions at one time. If initial proportion of each compartment changes, then quanta concentration as well as infected proportion can change accordingly.

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