Airborne infection R-numbers for regularly attended spaces:

COVID-19 a case-study

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Abstract

The risk of airborne infection for COVID-19 needs to be better understood and is especially urgent during the current pandemic. We present a method to determine the relative risk that can be readily deployed on either modelled or monitored CO\(_2\) data and occupancy levels within an indoor space. Moreover, for spaces regularly, or consistently, occupied by the same group of people, e.g. an open-plan office or a school classroom, we establish protocols to assess the absolute risk of airborne infection of this regular attendance at work or school. In so doing, we are able to calculate the
expected number of infections arising from a single regular attendee becoming infectious and remaining pre/asymptomatic, i.e. we present a robust methodology to calculate the absolute reproductive number of these spaces. We demonstrate our model by calculating risks for both a modelled open-plan office and by using monitored data recorded within a small naturally ventilated office. Results suggest that attendance at work is unlikely to significantly contribute to the pandemic if relatively quiet desk-based work is carried out in the presence of adequate ventilation. However, these spaces are likely to contribute significantly to the pandemic if ventilation is inadequate and/or activity levels increase.

1 Introduction

The novel coronavirus disease (COVID-19), which causes respiratory symptoms, was declared a pandemic by the World Health Organization (WHO) on the 11th March 2020 — thereby marking its global impact. Transmission of such respiratory infections occurs via virus-laden particles (in this case the virus SARS-CoV-2) formed in the respiratory tract of an infected person and spread to other humans, primarily, via three routes: the droplet route, the contact route and the airborne route (Mittal et al., 2020). After some initial resistance, and significant pressure from the scientific community (e.g. Morawska et al., 2020; Morawska & Milton, 2020), the WHO finally acknowledged the possibility of airborne infection for COVID-19 on the 8th July 2020 (The Independent, 2020). In our present article, we focus on assessing the risk of infection of respiratory diseases via the airborne route, taking COVID-19 as an example; ultimately, deriving a methodology for calculating a meaningful R-number for any indoor space that is regularly attended by the same group of people and any airborne disease for which the duration over which infectors remain pre/asymptomatic is known (within some bounds).

The pioneering work of Wells (1955) and that which followed by Riley et al. (1978) established methods, commonly referred to as the Wells-Riley model, for quantifying the
risk of airborne infection of respiratory diseases. However, the methodology was restricted
to indoor spaces which were in a steady-state with a known constant rate of ventilation 
of outdoor air. Rudnick & Milton (2003) greatly extended the practical application of the 
Wells-Riley model; removing the need to assume the indoor space was in steady-steady
and, crucially, negating the need to assess nor assume the rate of ventilation of outdoor
air – a notoriously difficult quantity to measure robustly (see Appendix B for a detailed
discussion). Rudnick & Milton (2003) achieved this via the realisation that the risk of
airborne infection could be directly inferred via measurements of CO₂ “if the airspace is
well mixed”. We generalise the model of Rudnick & Milton (2003) relaxing the assumption
of a well-mixed space and to further account for occupation profiles and activity levels which
vary in time.

We note that no single novel step of our analysis is particularly exceptional. We build
directly on the model of Rudnick & Milton (2003) and carry out analysis in the same vein as
others (e.g. Buonanno et al., 2020). For many airborne infections the likelihood of spread
within the vast majority of indoor spaces, even over periods of a few hours, is reasonably
low (as we show for the coronavirus SARS-CoV-2 and the resulting disease COVID-19).
However, our key realisation is that there exists a significant proportion of indoor spaces
which are, for a significant portion of each day, attended by the same/similar group of
people (e.g. open-plan offices and school classrooms), herein ‘regularly attend spaces’. Our
model enables the likelihood of the spread of infection to be calculated (from either easily
obtainable monitored data or modelled data) over multiple day-long durations. Hence, in
the case of COVID-19 (for which infectors are estimated to remain pre-asymptomatic for
5-7 days) our model calculates the likely number of people that become infected during a
period in which a pre/asymptomatic infector regularly attends the space.

We derive an extended airborne risk model in §2 assess the risk of infection in a
modelled open-plan office in §3 and use monitored data from naturally ventilated office in §4 to estimate the infection risk. Finally we draw conclusions in §5.

1.1 The Wells-Riley approach to airborne infection risk

The pioneering work of Riley et al. (1978) defined the infectivity rate as

\[ \lambda = \frac{I p q}{Q}, \]  

where \( I \) is the number of infected people, \( p \) is the breathing (pulmonary ventilation) rate, \( Q \) is the ventilation (outdoor air supply) rate, and \( q \) is the unit of infection, quantum (see Riley et al. 1978, for discussion), which varies significantly between disease, with activity level, and (as with all biologically derived parameters) with individual human beings. For many diseases and relevant activity levels, appropriate values of \( q \) have been determined and are reported in the literature – however, significant uncertainties are associated with these values. Moreover, the variability due to individuality is challenging to reflect, see §3 for more discussion. In particular, high values of risk are obtained from quanta generation rates derived from so called ‘superspreading events’ – we choose not to dwell on such cases but note that should we have done so then the risks reported herein would be dramatically increased (see, for example, table 1). For a given demographic and activity level within the space the breathing rate \( p \) can be taken as constant and values are widely reported in the literature, the number of infected people \( I \) is an input to the model usually taken to be constant.

Riley et al. (1978) was no doubt aware of the significant challenges in measuring, or even inferring, the outdoor air supply rate to a given indoor space (see Appendix B). Instead, it was chosen to report the model in a form that can only be applied to indoor spaces for which the air is relatively well-mixed and the flows within are in steady-state.
Under these restrictive assumptions the classical Wells-Riley equation is recovered, namely that the likelihood, $P$, that infection spreads within a given indoor space during a time interval $T$ is

$$P = 1 - \exp \left( -\frac{I p q T}{Q} \right). \tag{2}$$

2 A model for airborne infection risk in transient spaces with variable occupancy and activity levels

As the insightful work of Rudnick & Milton (2003) highlighted, airborne infection can only occur through the breathing of rebreathed air that is infected. Within most indoor spaces human breathing is the dominant source CO\(_2\) and so the fraction $f$ of rebreathed air can be inferred from the ratio of the CO\(_2\) concentration within the space (above outdoor levels, $C_0$) to the concentration of CO\(_2\) added to exhaled breath during breathing, $C_a$, giving

$$f = \frac{C - C_0}{C_a}, \tag{3}$$

where $C$ in the measured CO\(_2\) within the space. Denoting the total number of people within the space $n$ gives the fraction of rebreathed air that is infected as $fI/n$. Rudnick & Milton (2003) chose to express their result as

$$P = 1 - \exp \left( -\frac{I n q \int_0^T f \, dt}{Q} \right) = 1 - \exp \left( -\frac{I q f T}{n} \right). \tag{4}$$

As they point this result “has very general applicability; it is valid for both steady-state and non-steady-state conditions and when the outdoor air supply rate varies with time”. Furthermore, we highlight that their assumption of a well-mixed space was not entirely necessary, their result provides the likelihood that a person (at the same location as the CO\(_2\) sensor within the space) becomes infected assuming only that the infected and uninfected
rebreathed air are relatively well-mixed, and not that the air within the space is well-mixed.

We extend the generality of (4) with greater application in mind by noting that the likelihood of someone becoming infected within a given space can be determined from

\[ P = 1 - \exp\left(-\int_0^T \lambda \, dt\right) = 1 - \exp\left(-\int_0^T f_I \frac{I}{n} q \, dt\right). \]  

(5)

In so doing (5) extends the work of Rudnick & Milton (2003) to allow the airborne infection risk to be assessed within indoor spaces that also have variable occupancy and activity levels. For a derivation of (5) from first principals see Appendix A. The applicability of our extension will be highlighted throughout §4.

2.0.1 Quantifying the relative risk for changes in environmental management

To examine the effects of a particular change in conditions within a given indoor space, e.g. change in ventilation rate, occupancy level/behaviour, etc..., it is informative to define a ‘base case’ scenario for which the likelihood of infection during a time interval \( T \) is \( P_0 \) and quantify the airborne infection risk of chosen scenarios relative to the base case. Writing (5) as a Taylor series expansion, relative risk can be expressed as

\[ RR = \frac{P}{P_0} = q \frac{\int_0^T f_I \frac{I}{n} q \, dt - \left(\int_0^T f_I \frac{I}{n} q \, dt\right)^2}{\int_0^T f_I \frac{I}{n} q \, dt - \left(\int_0^T f_I \frac{I}{n} q_0 \, dt\right)^2} + \frac{\left(\int_0^T f_I \frac{I}{n} q \, dt\right)^3}{\int_0^T f_I \frac{I}{n} q_0 \, dt} + \ldots, \]  

(6)

where the subscript 0 refers to the values in the base case. For any given disease and any combination of rebreathed air fraction and occupancy level/activity profiles a time interval \( T = T_s \) can be selected over which the probability of infection is low, i.e. \( \int_0^{T_s} f \frac{I}{n} q \, dt \) is small, such that the relative risk can be accurately assessed by consideration of only the leading order terms. For a given disease and for scenarios in which human activity within the space remains broadly unchanged, the quanta generation rate can be considered
constant and \( q = q_0 \). In such cases, a good approximation for the relative risk is provided by

\[
RR_s = \frac{\int_0^{T_s} f \frac{L}{t} \, dt}{\int_0^{T_s} f_0 \frac{L_0}{t_0} \, dt}.
\] (7)

We note that any evaluation of \( RR_s \) will result in a conservative overestimate of the infection risk (since the highest order term neglected is negative, see (6)), the scale of any overestimate can be easily assessed. Crucially, \( RR_s \) is not dependent on the quanta generation rate – a notoriously difficult quantity to parameterise – and hence for airborne infection risk assessment \( RR_s \) can be reported noting the results are valid for all diseases (note that the duration \( T_s \) for which the approximation remains valid does change with disease). Moreover, these results can be reported with a greater degree of certainty.

2.0.2 Defining absolute risk and the R-number for a given indoor space

An indoor space can be considered as contributing to the spread of a disease if an infected person attends the space for a duration over which it is more likely than not that they infect others. In the case that someone is showing symptoms of the disease it is reasonable to assume that they cease attending the space or that they be required to do so. Individuals can remain infectious and asymptomatic/presymptomatic for time periods of multiple days (which we denote as \( T_A \)) and this renders (4) unsuitable for quantifying this likelihood for most indoor spaces. However, for regularly attended spaces e.g. open plan offices and school classrooms, the probability \( P_A \) that someone becomes infected via the airborne transmission route (assuming an infected person attends the space) can be robustly determined via our formulation (5). To do so, time series data for the rebreathed air fraction (monitored or modelled), the occupancy level and quanta generation rate are required over the duration \( T_A \). For a given disease, assuming the activity levels (per capita) remain broadly the same within the space, the quanta generation rate can be assumed constant. For real-
world assessment, $f$ and $n$ can be obtained from monitored CO$_2$ and occupancy data, respectively. Moreover, for model cases this can easily be calculated. We demonstrate examples of this for model building spaces ($\S$4.1), and using monitored data an existing open plan office ($\S$4.2) taking COVID-19 as a case study.

As elegantly pointed out by Rudnick & Milton (2003) their formulation (4) can be used to determine a basic reproductive number for an airborne infectious disease within an indoor space; this being the number of secondary infections that arise when an infectious individual is attending the space and everyone else is susceptible. The absolute reproductive number $R_A$, i.e. the number of people likely to become infected via the airborne route (which is the probability of someone becoming infected, given by (5), multiplied by the number of susceptible persons) is for regularly attended spaces

$$R_A = (N - 1) \left[ 1 - \exp \left( - \int_0^{T_A} f \frac{1}{n} q \, dt \right) \right],$$

where $N$ is the total number of people that regularly attend the space.

To summarise our modelling, we have developed practical statistics to assess airborne infection via relative risk based scenario testing ($RR$ and $RR_s$), the absolute probability of infection ($P_A$), and the absolute reproductive number of an indoor space ($R_A$). All of these can be calculated by obtaining/modelling representative CO$_2$ data. Moreover, for measured/modelled CO$_2$ distributions within the space, on assuming the infected & uninfected rebreathed air are mixed, these statistics can be calculated and their variation within the space investigated.
3 Determining appropriate quanta generation rates

As with all Wells-Riley based infection modelling an input parameter for which great uncertainty abounds is the quanta generation rate, \( q \). Given the uncertainty, we include results of scenario tests at various feasible levels of \( q \), all values of which are taken from the data of Buonanno et al. (2020). As a base case, which we deem appropriate for the regularly attended spaces on which we focus (namely, open-plan offices and class rooms) we take a value of \( q = 1 \) quanta/hr — this is obtained by taking \( c_x = c_i c_v \approx 7 \times 10^6 \) RNA/ml, where \( c_i = \{0.1, 0.01\} \) is the ratio between infectious quantum and the infectious dose expressed in viral RNA copies, and \( c_v = \{7 \times 10^7, 7 \times 10^8\} \) RNA/ml is the viral load measured in sputum. These values obtained by consideration that for most of the time, in most open-plan offices and classrooms, most of the occupants are sitting breathing with perhaps a small number vocalising — the data for whispered counting falls between these two activities and is rather more close to breathing — as such, for our base case, we take data for whispered counting from Buonanno et al. (2020) and use their results to map our selected values of \( c_x \) to values of quanta generation rates \( q \). Moreover, we consider a scenario in which the occupants within the open-plan office or classroom are (on average) all vocalising/talking (e.g. a call-centre or noisy classroom), taking again \( c_x \approx 7 \times 10^6 \) RNA/ml gives \( q \approx 5 \) quanta/hr. In addition, we consider a scenario in which the viral load in sputum is somewhat reduced, i.e. \( c_v \approx \{2 \times 10^7, 2 \times 10^8\} \) RNA/ml, giving \( q \approx 0.3 \) quanta/hr.

4 Results using COVID-19 as an example

We present results for a modelled open-plan office and for data monitoring both CO\(_2\) and occupants in a small naturally ventilated office. Regarding the presence of infectors, given that we assume occupants arrive and leave over realistic periods of time (i.e. they do not all arrive and leave at once), there exists at least two reasonable choices. One
could (conservatively) assume that the infector is always the first to arrive and the last to leave. Alternatively, one could assume that there is always a constant proportion of the (current) occupants infected such that when the space is occupied to design capacity there is a single infector (this results in the number of infectors, $I$, taking non-integer values outside design occupancy which is inconsequential). Should one choose the former option, there is potential that higher risks are reported for more sparsely occupied spaces (since by enforcing a single infector the rebreathed air is more concentrated with infectious particles) or equivalently by allowing more occupants one risks perceiving the space as less risky since infected breath is assumed more dilute — in the absence of knowledge as to who is infected, this cannot be reasonable for comparison of different occupancy profiles. As such, we choose to present results for this latter choice and we note that (for the occupancy profiles examined) the estimated risks would be approximately 20% higher if the more conservative choice were made.

4.1 Application to a model open-plan office

By way of example, we first consider a moderately sized open-plan office, of floor area 400 m$^2$ and floor-to-ceiling height 3.5 m, which is designed to be occupied by 40 people (in accordance with the typical occupancy densities suggested in CIBSE, 2006). We assume that occupants arrive steadily between 08:00 and 09:00 each morning, each take a one hour lunch break during which they leave the office, and leave steadily between 17:00 and 18:00 each day. While within the office we assume that (on average) each occupant breathes at a rate of approximately $p = 8$L/min with a CO$_2$ production rate of 0.3$L$/min, giving $C_a = 0.038$ and we take the outdoor CO$_2$ level to be 400 ppm (Rudnick & Milton, 2003).

Our model run for this open-plan office gives, for the base case, the absolute risk of infection during a period of pre/asymptomatic COVID-19 infection as $P_A = 1.1\%$. If one had have taken the classical Wells-Riley model (13), and taken $T$ to be the simple sum
of occupied hours, the level of risk reported would have been $P = 1.3\%$. Hence, the risks reported would have again been around 20% higher. We note the key benefit of our model is the ability to use monitored CO$_2$ and occupancy data as we show in §4.2.

4.1.1 The impact of varied quanta generation rates

We first examine the impact of varied quanta generation levels; namely, $q = \{1, 0.3, 5\}$ (see §1 for a full discussion). Figure 1 plots the absolute likelihood that someone becomes infected within the office over the period which an infector is expected to remain pre/asymptomatic, i.e., 5 working days (since the period of pre/asymptomatic infectivity for COVID-19 is estimated as 5–7 days). The plot shows that in the base case the absolute risk, $P_A$, of infection within this open-plan office is just over 1%, assuming a lower viral load is appropriate the risk drops to around $P_A = 0.3\%$. However, if the open-plan were a call-centre then this risk that someone becomes infection through attending work increases to above 5%.

One can, of course, examine the relative risk; however, as expected from (6) the results are broadly constant in time, taking an initial value of $RR = q/q_0$ (see(6)), and then remain dominated by the ratio of scenarios quanta. For example, in this office at the end of a pre/asymptomatic period, examining the scenario that the open-plan office becomes equivalent to a call-center gives $RR_A = 4.9$, and assuming the appropriate viral load for the disease is lower gives $RR_A = 0.3$. For the sake of useful approximation, if one includes only the first order terms in the risk then the relative risks are simply 5.0 and 0.3, respectively (since we take $q/q_0 = 5$ and $q/q_0 = 0.3$, respectively see (6) cf. (7)).

4.1.2 The importance of ventilation/outdoor air supply rates

The qualitative increase in the absolute risk during a period of pre/asymptomatic infection with varied outdoor air supply rate, $Q$, is broadly similar to that shown in figure 1 with the
Figure 1: The variation in the likelihood of infection with time over the five day pre/asymptomatic period. The different curves highlight the change in risk with assumed quanta generation rate: $q = 1$ quanta/hr (red), $q = 0.3$ quanta/hr (blue), and $q = 5$ quanta/hr (black).
Figure 2: The variation in the relative risk, $RR$, of infection with time over the five day pre/asymptomatic period. The different curves highlight different scenarios, namely: the base case, $Q = 10 l/s/p$ (red), increased vent, $Q = 20 l/s/p$ (blue), and decreased vent, $Q = 4 l/s/p$ (black).
base case (of course) again being \( P_A = 1.1\% \). Doubling the ventilation rate to \( Q = 201/s/p \) gives \( P_A = 0.6\% \), and decreasing the outdoor air supply rate to \( Q = 41/s/p \) results in \( P_A = 2.2\% \) (see table \( \text{II} \)). It is interesting to note that, in the case of examining scenarios of changing ventilation rates then the relative risk \( RR \) takes an initial value of unity and it is only over time that the ventilation works to alter the accumulation of infected re-breathed air within the space. In this open-plan office the relative risk reaches an approximately steady value of \( RR \approx 0.55 \) after around 6 hours in the case that the ventilation is doubled, and in the case of decreased ventilation \( (Q/Q_0 = 0.4) \) then \( RR \approx 2.0 \) is reached after approximately 10 hours. As occupancy alters within the office each time, and the fraction of infected rebreathed air increases/decreases at different rates, the relative risk deviates (temporarily) from its steady-state value (these deviations are more pronounced in the case the ventilation is reduced, black curve in figure \( \text{2} \)).

4.1.3 The R-number for an open-plan office

We run our model for the R-number \( (8) \) for a period of pre/asymptomatic infectivity (5–7 days) varying both the quanta generation rate \( (q = \{0.3, 1.0, 5.0\} \text{ quanta/hr}) \) and the outdoor air supply rate \( (Q = \{4, 10, 20\} \text{ l/s/p}) \). The results are presented in table \( \text{II} \) and confirm that, for this office, in most cases it is unlikely that an employees attendance at work will significantly contribute to the spread of COVID-19. The said, if the RNA copies/viral

| R-numbers, \( R_A \) | \( Q = 41/s/p \) | \( Q = 101/s/p \) | \( Q = 201/s/p \) |
|----------------------|-----------|---------------|---------------|
| \( q = 0.3 \text{ quanta/hr} \) | 0.25 | 0.13 | 0.07 |
| \( q = 1.0 \text{ quanta/hr} \) | 0.84 | 0.42 | 0.24 |
| \( q = 5.0 \text{ quanta/hr} \) | 4.0 | 2.1 | 1.2 |
| \( q = 20 \text{ quanta/hr} \) | 14 | 7.6 | 4.4 |
| \( q = 100 \text{ quanta/hr} \) | 35 | 26 | 18 |

Table 1: COVID-19 R-numbers, \( R_A \), for an open-place office (floor plan of 400 m\(^2\) and floor-to-ceiling height of 3.5 m) occupied by 40 people for 8 hrs each day over the period that a pre/asymptomatic person remains attending work.
load are as expected by Buonanno et al. (2020) and the office is poorly ventilated then R-number for this office may hover dangerously close to unity. Moreover, if used for particularly vocal activities, e.g. a call-center or sales office, then these office spaces have the potential to significantly contribute to the spread of COVID-19 with R-numbers of nearly 8 being realised for offices with appropriately designed ventilation.

To end this section, we note that Buonanno et al. (2020) report far higher quanta generation rates for ‘superspreaders’ (which maybe some combination of the particular activity being undertaken, the environment quality, and the biological response of an individual). In table 1, the last two lines reports the R-numbers for our office appropriate superspreaders the results are worrisome with a single infector resulting in approximately 30 new infections.

4.2 Airborne infection risk from monitored data in open plan offices

To demonstrate the application of our model to indoor spaces with monitored CO₂ and occupancy data we were provided access to data recorded by the ‘Managing Air for Green Inner Cities (MAGIC)’ project (http://www.magic-air.uk). The data were recorded in a small office which had a design capacity of eight people, although during the times for which we were provided data never more than six people attended the office. The office is naturally ventilated with openable sash windows on opposite sides of the building. The floor area is approximately 37.6 m² and the floor-to-ceiling height is 2.7 m; Song et al. (2018) provide full details of the monitored space and the monitoring equipment used but it should be noted the monitored office is not of a modern design and is not well-sealed nor well-insulated.
Figure 3: The intra-day variation in occupancy (upper panes, a) and b)), monitored CO₂ (middle panes, c) and d)) and the corresponding risk of airborne spread of COVID-19 (lower panes, e) and f)) during 29th Sep 2017 (left-hand panes, a), c) and e)) and 5th Oct 2017 (the right-hand panes, b), d) and f)). Data are plotted from six CO₂ monitors placed at various locations and heights (between 73 cm and 242 cm from the floor). On the 29th Sep (left-hand panes) windows on opposite sides of the room were opened (creating an opened area of around 0.24 m²) from 08:00 until 20:00 whilst on the 5th Oct (right-hand panes) the windows remained closed all day.
4.2.1 The role of opening windows in reducing risk

Figure 3 a) and b) show the occupancy profiles during two days in 2017; namely, 29th Sep and 5th Oct, respectively. During 29th Sep the windows were opened on both sides of the building (providing an opened area of 0.24 m²) at around 09:00 and remained so until after 20:00; whilst on 5th Oct the windows remained closed all day and we note that the spike in CO₂ at around 16:15 on this day corresponds to a brief visit during which 22 people were in the office. The monitored CO₂ profiles (figure 3 c) and d)) were obtained at six locations of differing heights (between 73 cm and 242 cm from the floor) and positions within the office. It is most striking that the CO₂ levels are markedly higher on 5th Oct when the windows remained closed. Crucially, these elevated CO₂ levels translate into increased risk of airborne infection for the occupants – in this case the risk of infection being approximately doubled on the day when the windows remained shut. In addition, at times (e.g. between about 14:00 and 17:00 on 25th Sep) there is a marked variation in measured CO₂ levels dependent on location. It follows that this variation in CO₂ is reflected in the infection risk levels which shows that the location within the office one was breathing affected the risk of infection by around 20% on the day the windows were closed and a much more substantial variation on the day the windows were open.

Figure 4 a) shows the occupancy data for the monitored office over a five day period in September 2017. During this five day period the office windows were open for some significant portion of each day. The accompanying monitored CO₂ data is shown in figure 4 b) and it should be noted that data are missing between 13:30 and 19:00 on 27th September. The risk of airborne infection for COVID-19 is shown in the lower-pane with the risk rising gradually over the period of pre/asymptomatic infectivity reaching an absolute risk 0.059 ≤ \( P_A \) ≤ 0.064 depending on where within the office the occupant would have been located. The R-number for the monitored office over this period is 0.3 ≤ \( R_A \) ≤ 0.32 – reassuringly
Figure 4: The variation in a) occupancy, b) monitored CO₂, and c) the corresponding risk of airborne spread of COVID-19, over a period of pre/asymptomatic occupancy of the monitored office. Data are plotted from six CO₂ monitors placed at various locations and heights (between 73 cm and 242 cm from the floor).
below unity and indicating that this naturally ventilated office was likely to have been receiving somewhere between $101/s/p$ and $201/s/p$ (see table). From examination of the monitored office during periods when the windows were closed the R-number might approximately double to $R_A \approx 0.6$. We note however, that this the R-number is likely to be considerably higher for more modern well-sealed offices.

5 Conclusion

Taking COVID-19 as an example we have derived a simple model to estimate the likelihood of airborne infection within indoor spaces which allows for occupancy levels and behaviour which vary in time. Our model requires only monitored or modelled data for CO$_2$ and occupancy levels and behaviour combined with some estimates of appropriate quanta generation rates. We demonstrated results taking a modelled office and also using monitored data. We are able to conclude that for regularly attended indoor spaces that have ventilation provision in-line with guidance (e.g. CIBSE, 2006) attendance of work or school is unlikely to significantly contribute to the spread of COVID-19. However, should these spaces be poorly ventilated (e.g. 41/p/s) then the R-number associated with the space should be expected to be close to unity. For even adequately ventilated spaces if the occupants are very vocal (e.g. a call-centre or noisy classroom) then one should expect around two new COVID-19 infections for every single infector. In addition, we note that we chose not to focus on superspreaders, but instead considered conditions more in-line with the medium of the population. Should we have chosen to examine superspreaders then our results would have been more alarming with regularly attended spaces giving rise to between 5 and 35 new infections from every infector.

We strongly recommend that monitoring of CO$_2$ is carried out for indoor spaces. Where these spaces can be considered to broadly conform to our definition of a regularly attended space then we further recommend that occupancy profiles are recorded. In so doing, we
provide a simple methodology for those responsible to calculate a meaningful R-number for airborne infection (for COVID-19) within their indoor space. Irrespective of this, we believe that consideration of the relative rate of increase in infection risk should be considered for all indoor spaces; this can be expressed (see appendix A) as $\lambda = \frac{(C - C_0)}{C_a n}$. For a given disease and activity level it is worth noting that $\lambda \propto \frac{(C - C_0)}{n}$. As such, one can simply infer the rate at which the likelihood of infection is increasing by consideration of the ratio of excess CO$_2$ and the current number of occupants.

Finally, we conclude that the risk of COVID-19 being spread by the airborne route is not insignificant and varies widely with activity level and environmental conditions which are predominantly determined by the bulk supply of outdoor air. We hope that our contribution proves to be illuminating both for assessing the airborne infection risks within regularly attended spaces and for consideration, and ideally monitoring, of these risks in more generic indoor spaces.

### A Modelling from first principles

In order to demonstrate the underlying assumptions and highlight the limits of applicability we revisit the formulation of the Wells-Riley equation ([Wells, 1955; Riley et al., 1978]). We wish to determine the likelihood, $P$, that infection spreads within a given indoor space during a time interval $T$. Denoting the probability that no one becomes infected during this time $P(0, T)$ gives that

$$P = 1 - P(0, T).$$  \hspace{1cm} (9)

The number of infected people, $I$, is discrete (i.e. an integer) and since time is continuous we can consider a small period of time, $\delta t$, during which either no one becomes infected or one person becomes infected. Defining the infectivity rate $\lambda$ (see below for a detailed discussion) gives the likelihood that one person becomes infected during this small time
period as

\[ P(1, \delta t) = \lambda \delta t. \]  

(10)

Assuming that each infection occurs independently of the last

\[ P(0, t + \delta t) = P(0, t)[1 - P(1, \delta t)] = P(0, t)[1 - \lambda \delta t]. \]  

(11)

Rearranging and taking the limit \( \delta t \to 0 \) gives

\[ \frac{P(0, t + \delta t) - P(0, t)}{\delta t} = \frac{dP(0, t)}{dt} = -\lambda P(0, t), \]  

(12)

integrating and substituting into (9) gives

\[ P = 1 - \exp\left(-\int_0^T \lambda dt\right). \]  

(13)

Hence the likelihood \( P \) can be evaluated for any known functional form of \( \lambda \), crucially, as we go on to demonstrate, this can inferred from data measured within indoor spaces which records occupancy level profiles and CO\(_2\) concentrations.

B The challenges of measuring ventilation rates or inferring ventilation rates from monitored CO\(_2\)

The original formulation of the Wells-Riley equation requires parameterisation of not only the quanta generation rate, \( q \), but also evaluation of the infectivity rate which in the general case must be evaluated via the integral

\[ \int_0^T \frac{I p q}{Q} dt. \]  

(14)
Assuming broadly constant activity levels within a space the breathing rate, $p$, and (for a given disease) the quanta generation rate can be regarded as time independent. Furthermore it may be reasonable to assume the number of infected people within space remains unchanged if one is examining the likelihood of spread. Moreover, if the ventilation rate $Q$ can be assumed constant and if the space is in steady-state then the probability of infection occurring during the period $T$ can be simply expressed as

$$P = 1 - \exp \left( \frac{I p q}{Q} T \right).$$

(15)

However, it is this last assumption that is most troubling since it is only reasonable if the all connections to the space (windows, doors, vents, etc...) are sealed over a time exceeding the transient ventilation effects (which typically remain significant for multiple hours), infiltration rates remain constant or negligible, and the ventilation system supplies outdoor air at rates which are insensitive to changes in the pressure differences between indoors and outdoors that arise due to changes in temperature and wind — this makes application of (15) difficult. As we will discuss, measuring or inferring the ventilation rate within an operational occupied space is non trivial and hence evaluating (14) is impractical. The insightful work of Rudnick & Milton (2003) solved many of these challenges in assessing airborne infection risk and forms the basis for the modelling described in §2.

The magnitude of infection risk changes with the seasons for numerous viral infections (including influenza) and these may arise for a variety of factors. These might include: changes in the viability of the virus if typical temperatures and/or humidity of indoor environments vary with the season, or if changing levels of natural UV light are significant and effect viability; changes in behaviour, for example, staying indoors more during colder seasons; changes due to the seasonal variations that occur in immunity (Dopico et al., 2015); and, crucially for the airborne transmission route changes in ventilation (i.e. outdoor
air supply) rates that occur as moderated indoor temperatures are demanded once outdoor temperatures vary with the season. We assert that this last factor, changing outdoor air supply rates, is highly significant yet it is poorly evidenced.

Outdoor air may enter an indoor space via a ventilation system, windows, doors, vents, cracks in the building fabric or, indeed, though the very fabric itself (i.e. many building materials, e.g. bricks, are porous). As such, there is significant scope for both intentional and unintended supply of outdoor air. Directly measuring the air flow through all of the potential pathways for any given indoor space in impractical. Pressure testing can be used to measure infiltration rates but cannot assess the ventilation rates in operational settings.

Indoors, human activity is typically the major source of CO$_2$ while outdoor CO$_2$ levels remain broadly constant. Therefore, if the rate of CO$_2$ production from human activity within a space can be estimated, CO$_2$ provides a suitable proxy from which to attempt inference of the ventilation/outdoor air supply rate within the space. Consider an indoor space in which both occupancy levels and CO$_2$ are monitored. If the activity levels of individuals remains broadly similar and the CO$_2$ monitored, $C$, (only point measurements are practically possible) can be regarded as indicative of the CO$_2$ levels throughout the space, i.e. the CO$_2$ is relatively well-mixed within the indoor air, then since CO$_2$ is inert so its conservation requires that

$$V \frac{dC}{dt} = npC_a - Q(C - C_0),$$  \hspace{1cm} (16)

where $n$ is the number of people in the space and $C_a$ is the volume fraction of CO$_2$ added to exhaled breath during breathing. As discussed, it is unwise to regard the ventilation rate as constant for most indoor spaces, i.e. $Q = Q(t)$, and this renders (16) non trivial to
integrate analytically. Thus if one wishes to examine the ventilation rate

\[ Q = n p \left( \frac{C_a}{C} - \frac{V}{C} \frac{dC}{dt} \right), \]  

(17)
can be evaluated with monitored occupancy and CO\textsubscript{2} data. However, as with all real-world data, the CO\textsubscript{2} signal is likely to contain some non-negligible level of noise and the dependence on \( \frac{dC}{dt} \) will, in most cases, render evaluation of ventilation/outdoor air supply rate via (17) unsuitable.

An alternate approach is to examine the monitored occupancy data to determine the time at which the room becomes unoccupied. Assuming that the ventilation rate remains unchanged thereafter (which will only be the case if ventilation systems are left operational, and any changes in the ventilation/outdoor air supply rate due to the effects of wind and temperature variations are negligible) the CO\textsubscript{2} concentration within the space, assuming the air within remains relatively well-mixed, will decay exponentially. By exponential fitting to the monitored data during this period a ventilation/outdoor air supply rate can be inferred. However, curve fitting to real-world data is prone to variability due to choices of the input parameters (e.g. the period over which exponential decay to determine to be observed) and subject to influence from noise within the data, thereby rendering the results unreliable. Moreover, this process is hard to automate and so typically requires significant manual intervention, making it unsuitable for the analysis of large data sets.

In summary, direct or inferred measurements of ventilation/outdoor air supply rates are extremely challenging. For this reason, and those described in §2, we council that to assess airborne infection risks no attempts be made to directly assess outdoor air supply/ventilation rates to indoor spaces. Instead, we suggest widespread monitoring of CO\textsubscript{2} within spaces combined with measured/estimated occupancy profiles, which with applica-
tion of our extensions (§2) to the work of Rudnick & Milton (2003) can be used to directly assess the airborne infection risk within a given space. Where required, simple modelling can be carried out to inform and assess practical mitigation strategies.
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Competing interests

The authors declare no competing interests.