
AVOIDING COVID-19: AEROSOL GUIDELINES

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ABSTRACT

The COVID-19 pandemic has brought into sharp focus the need to understand respiratory virus transmission mechanisms. In preparation for an anticipated influenza pandemic, a substantial body of literature has developed over the last few decades showing that the short-range aerosol route is an important, though often neglected transmission path. We develop a simple mathematical model for COVID-19 transmission via aerosols, apply it to known outbreaks, and present quantitative guidelines for ventilation and occupancy in the workplace.

Keywords SARS-CoV-2 · COVID-19 · coronavirus

1 Introduction

The world is learning to navigate the COVID-19 pandemic and a great deal of information about the disease is already available [1, 2, 3]. In order to adjust to this new reality it is important to understand what can be done to avoid infection, and to avoid infecting others.

SARS-CoV-2, the coronavirus which causes COVID-19, is thought to be transmitted via droplets, surface contamination and aerosols [4, 5, 6, 7, 8, 9]. Contact tracing of COVID-19 outbreaks points to aerosols as a possibly dominant mechanism of transmission since most outbreaks occur in closed environments, while they are rare in environments which disfavor aerosol transmission (e.g., outdoors) [10]. Furthermore, infection via inhalation of aerosols and small droplets dominates large droplet exposure in most cases for physical reasons linked to complex, but well understood fluid dynamics [11, 12, 13, 14]. Despite this fact, there is a common misconception that aerosol transmission implies efficient long-range transmission (as in measles), and thus that the absence of long-range transmission implies an absence of aerosol transmission. The truth is more nuanced, and includes the possibility of a dominant short-range aerosol path limited by pathogen dilution, deposition, and decay [15].

The production of aerosolized viruses by a contagious individual occurs naturally as a result of discrete events (e.g., coughing or sneezing), or through a continuous process like breathing or talking [16]. Droplets which are formed with diameters less than $\sim 50 \mu\text{m}$ quickly lose most of their water to evaporation, shrinking by a factor of 2-3

in diameter and becoming “droplet nuclei”. These fine particles settle very slowly and mix with the air in the environment [17].

Aerosol transmission happens when a susceptible individual inhales these now sub- $20 \mu\text{m}$ droplet nuclei that are suspended in the air around them [18, 19]. This is thought to be the dominant transmission mechanism for influenza and rhinovirus [18, 20] and possibly also for SARS and MERS [21]. For influenza, it has been shown that aerosol particles as small as $1.5 \mu\text{m}$ are sufficient for transmission [22]. Furthermore, it is known from other viral respiratory diseases that aerosol exposure can result in infection and illness at much lower doses than other means (e.g., nasal inoculation) [23].

In line with current recommendations [24], we will assume that hand-hygiene protocols are being followed sufficiently well to ensure that surface contamination is sub-dominant. We will also assume that contagious individuals are wearing some kind of face covering which is sufficient to disrupt the momentum of any outgoing airflow [25] and catch large droplets [26]. These actions leave aerosols, investigated here, as the dominant transmission mechanism.

In the following sections we present a simple model for aerosol transmission, apply this model to known outbreaks, and develop guidelines for reducing the probability of transmission in the workplace. We start with a description of our infection model, followed by aerosol concentration models for a variety of situations, mitigation measures, a description of model parameters, and finally a discussion of risk assessment.

2 Infection Model

The probability of developing an infection P_{inf} given exposure to a volume V of saliva with a concentration ρ of virions is

$$P_{\text{inf}} = 1 - e^{-\rho V/N_{\text{inf}}} \quad (1)$$

where the infectivity N_{inf} is the number of virions needed to make infection likely in the lungs for aerosol inhalation [23, 27]. The infectivity value N_{inf} includes probable deposition location (i.e., upper vs. lower respiratory tract) and local deposition efficiency (e.g., not all inhaled droplets are deposited in the lungs [23, 28]). It should be noted, however, that small doses are less likely to cause *illness* than what is indicated by Eqn. 1 [23, 29].

Rather than work explicitly with the probability of infection P_{inf} , we will use the product of the viral dose and the infectivity

$$D_{\text{inf}} = \rho V/N_{\text{inf}} \quad (2)$$

as a proxy, and refer to it as the “infective dose”. Note that the infectivity and the viral concentration always appear together here, so only their ratio ρ/N_{inf} is important in our model. While ρ has been measured for COVID-19 [30, 31], N_{inf} has not been directly measured. We estimate N_{inf} from influenza and other coronaviruses [27], and check this value by applying our model to known outbreaks (see appendix A).

3 Aerosol Infective Dose Model

In this section we compute the infective dose which results from the presence of a contagious individual in various scenarios. This informs prescriptions for the maximum time a susceptible individual may be exposed to potentially contaminated air, or the minimum time between occupants in the same space. Each of these calculations uses the values in Table 1 and concludes with an exposure time limit for an infectious dose less than $D_{\text{inf}}^{\text{max}} = 0.1$ such that the accumulation of 4 such does results in a 10% probability of infection (see section 6).

3.1 Steady State in a Room

A contagious individual will shed virions into the room they occupy through breathing, talking, coughing, etc. The aerosolized virion concentration in a room can be expressed in the form of a differential equation as

$$\frac{d\rho_A(t)}{dt} = \rho_0 \frac{r_{\text{src}}}{V_{\text{room}}} - \rho_A(t) \left(\frac{1}{\tau_{\text{room}}} + \frac{1}{\tau_a} \right) \quad (3)$$

where ρ_0 is the nominal viral concentration in saliva, which is emitted in aerosol form at a rate of r_{src} , and τ_a is the viral decay time in aerosol form. The air cycle-time in the room is

$$\tau_{\text{room}} = V_{\text{room}}/r_{\text{room}} \quad (4)$$

where r_{room} is rate at which air is removed from the room (or filtered locally). The steady-state concentration is

$$\rho_A^{\text{SS}} = \rho_0 \frac{r_{\text{src}}}{V_{\text{room}}} \frac{\tau_{\text{room}}\tau_a}{\tau_{\text{room}} + \tau_a}. \quad (5)$$

and has units of viral copies per liter of air.

Breathing air in a room with an aerosol virus concentration ρ_A will cause an accumulation of exposure N_A (number virions) proportional to the time passed in that room t

$$N_A = \rho_A r_b t \quad (6)$$

such that

$$D_{\text{inf}}^{\text{SS}} = \frac{\rho_0}{N_{\text{inf}}} \frac{r_{\text{src}}}{V_{\text{room}}} \frac{\tau_{\text{room}}\tau_a}{\tau_{\text{room}} + \tau_a} r_b t. \quad (7)$$

If the room is well ventilated (i.e., $\tau_{\text{room}} \ll \tau_a$) the infective dose is approximately

$$D_{\text{inf}}^{\text{SS}} \simeq 0.01 \frac{r_{\text{src}}}{1 \text{ nL/min}} \frac{1 \text{ m}^3/\text{min}}{r_{\text{room}}} \frac{t}{1 \text{ min}} \quad (8)$$

for an office, lab or bathroom occupied by a contagious individual.

The infective dose crosses our example event exposure threshold of $D_{\text{inf}}^{\text{max}} = 0.1$ in an office-like space ($r_{\text{room}} \sim 10 \text{ m}^3/\text{min} \sim 350 \text{ cfm}$) after 100 minutes,

$$t_{\text{max}}^{\text{Room}} \simeq 100 \text{ min} \frac{1 \text{ nL/min}}{r_{\text{src}}} \frac{r_{\text{room}}}{10 \text{ m}^3/\text{min}}, \quad (9)$$

while in small spaces with less ventilation (e.g., a car or bathroom with $r_{\text{room}} \sim 1 \text{ m}^3/\text{min}$) the dose would cross the threshold after only 10 minutes. We have assumed that the contagious individual is quietly working such that $r_{\text{src}} = V_b$, but if they are talking (e.g., in video conference), or coughing occasionally, the source term may be higher by an order of magnitude or more (e.g., $r_{\text{src}} \gtrsim V_t$).

3.2 Steady State in a Building

When a susceptible individual occupies a room that is connected via the HVAC system to a room occupied by a contagious individual, there is the possibility of aerosols passing through the HVAC system [7, 38].

However, the volume of a droplet is proportional to its diameter *cubed*, which leads to the majority of the viral dose being delivered in the $\sim 10 \mu\text{m}$ droplet nuclei [17, 22], which are filtered by HVAC systems. Most HVAC filters will remove the majority of the viral load associated with COVID-19 transmission, and a high quality filter (i.e., MERV 12) is sufficient to remove $> 90\%$ of the larger droplet nuclei [39]. The remaining aerosols are further diluted by fresh make-up air, and then spread among all of the air spaces associated with the HVAC system. The associated infective dose in rooms connected to a room occupied by a contagious individual will be roughly

$$D_{\text{inf}}^{\text{Sys}} = (1 - f_{\text{sys}}) \frac{\rho_0}{N_{\text{inf}}} \frac{r_{\text{src}}}{V_{\text{sys}}} \frac{\tau_{\text{sys}}\tau_a}{\tau_{\text{sys}} + f_{\text{sys}}\tau_a} r_b t \quad (10)$$

where f_{sys} is the fraction of the viral dose which is removed by the filters or displaced by make-up air, and $\tau_{\text{sys}} = r_{\text{sys}}/V_{\text{sys}}$ is the time required to cycle the entire air volume through the HVAC system.

Description	Symbol	Value	Reference
Viral Load in Saliva	ρ_0	1000 /nL	[30, 31, 32]
Sneeze Aerosol Volume	V_s	1 μ L	[33, 34]
Cough Aerosol Volume	V_c	100 nL	[33, 34]
Talking Aerosol Volume	V_t	10 nL/minute	[33, 34]
Breathing Aerosol Volume	V_b	1 nL/minute	[33, 34]
Aerosol Decay Time	τ_a	2 h	[35, 36]
Breathing Rate	r_b	10 L/minute	[37]
Respiratory Infectivity	N_{inf}	1000	[27, 23]
Maximum Exposure and Minimum Wait Times			
Room Steady State	$t_{\text{max}}^{\text{Room}} \simeq 100 \text{ min}$	$\frac{1 \text{ nL/min}}{r_{\text{src}}} \frac{r_{\text{room}}}{10 \text{ m}^3/\text{min}}$	Eqn. 9
System Steady State	$t_{\text{max}}^{\text{Sys}} \simeq 100 \text{ min}$	$\frac{0.1}{1-f_{\text{sys}}} \frac{10 \text{ nL/min}}{r_{\text{src}}} \frac{f_{\text{sys}} r_{\text{sys}}}{100 \text{ m}^3/\text{min}}$	Eqn. 12
Occupancy Wait Time	$t_{\text{min}}^{\text{TE}} \simeq \tau_{\text{room}} \ln \left(100 \frac{V_{\text{src}}}{1 \mu\text{L}} \frac{1 \text{ m}^3/\text{min}}{r_{\text{room}}} \right)$		Eqn. 15
Passage Wait Time	$t_{\text{min}}^{\text{PS}} \simeq \tau_{\text{room}} \ln \left(100 \frac{V_{\text{src}}}{1 \mu\text{L}} \frac{10 \text{ m}^3}{V_{\text{room}}} \frac{\Delta t}{1 \text{ min}} \right)$		Eqn. 17
Flow Rate Conversion	$1 \text{ m}^3/\text{min} \simeq 35 \text{ cfm}$		

Table 1: Parameters used in the aerosol transmission model. See section 5 for more information.

In the well ventilated limit, as above, the infective dose is

$$D_{\text{inf}}^{\text{Sys}} \simeq 10^{-4} \frac{1-f_{\text{sys}}}{0.1} \frac{100 \text{ m}^3/\text{min}}{f_{\text{sys}} r_{\text{sys}}} \frac{r_{\text{src}} t}{10 \text{ nL}} \quad (11)$$

and the maximum occupancy time for $D_{\text{inf}}^{\text{Sys}} < 0.01$ is

$$t_{\text{max}}^{\text{Sys}} \simeq 100 \text{ min} \frac{0.1}{1-f_{\text{sys}}} \frac{10 \text{ nL}/\text{min}}{r_{\text{src}}} \frac{f_{\text{sys}} r_{\text{sys}}}{100 \text{ m}^3/\text{min}}. \quad (12)$$

We have set the r_{src} scale at 10 nL/min to allow for talking and an occasional coughs from the contagious individual. The maximum dose used in this example is 0.01 instead of 0.1 due to the likely larger cohort exposed (see section 6).

To understand why transmission through HVAC systems appears to be rare we note that even medium quality air filters (MERV 9) provide better than 99% filtering after some loading [39]. The value we use here assumes little or no fresh make-up air and is representative of an unloaded (i.e., clean) air filter, which is the *worst case*. Also, in buildings where the HVAC system does not recirculate air this type of transmission cannot occur (i.e., $f_{\text{sys}} = 1$).

3.3 Transient Occupancy and Events

Some spaces are occupied briefly by many people (e.g., bathrooms) and many not have time to come to steady state. Coughing and sneezing events can also cause a transient increase in the viral concentration in a room. The viral dose associated with these types of transients can be quantified as

$$D_{\text{inf}}^{\text{TE}} = \frac{\rho_0}{N_{\text{inf}}} \frac{V_{\text{src}}}{V_{\text{room}}} r_b \int_{t_1}^{t_2} e^{-t \frac{\tau_{\text{room}} + \tau_a}{\tau_{\text{room}} \tau_a}} dt \quad (13)$$

where the transient event occurs at time $t = 0$ and exposure is from time t_1 to t_2 .

We can estimate when a room is “safe” for a new occupant by computing the dose in the limit of $t_2 \rightarrow \infty$,

$$D_{\text{inf}}^{\text{TE}} \simeq \frac{\rho_0}{N_{\text{inf}}} \frac{V_{\text{src}}}{V_{\text{room}}} \tau_{\text{room}} r_b e^{-t_1/\tau_{\text{room}}} \quad (14)$$

where we have again assumed that $\tau_{\text{room}} \ll \tau_a$. This leads to a minimum wait time of

$$t_{\text{min}}^{\text{TE}} \simeq \tau_{\text{room}} \ln \left(100 \frac{V_{\text{src}}}{1 \mu\text{L}} \frac{1 \text{ m}^3/\text{min}}{r_{\text{room}}} \right) \quad (15)$$

where we have set the V_{src} scale at 1 μ L to allow for some coughing or a sneeze from the previous occupant.

As a concrete example, for a 70 square-foot bathroom with a 70 cfm fan in operation (i.e., $\tau_{\text{room}} \simeq 10$ minutes and $r_{\text{room}} = 2 \text{ m}^3/\text{min}$), Eqn. 15 gives a 40 minute wait time between occupants.

Going back to Eqn. 13, we can also compute the exposure due to brief passage through a common space in which $\Delta t = t_2 - t_1 \ll \tau_{\text{room}}$ (e.g., a halls, stairwells and elevators),

$$D_{\text{inf}}^{\text{PS}} \simeq \frac{\rho_0}{N_{\text{inf}}} \frac{V_{\text{src}}}{V_{\text{room}}} r_b \Delta t e^{-t_1/\tau_{\text{room}}}. \quad (16)$$

The minimum time between occupants for $D_{\text{inf}}^{\text{PS}} < 0.01$ is

$$t_{\text{min}}^{\text{PS}} \simeq \tau_{\text{room}} \ln \left(100 \frac{V_{\text{src}}}{1 \mu\text{L}} \frac{10 \text{ m}^3}{V_{\text{room}}} \frac{\Delta t}{1 \text{ min}} \right), \quad (17)$$

which will give negative values for large spaces or short passage times, indicating that no wait is necessary. However, this assumes well mixed air, so some mixing time is necessary to avoid a “close encounter” as described in the next section. For small spaces with poor circulation (e.g., elevators), on the other hand, long wait times are required.

3.4 Close Encounters

Mixing of aerosols into an airspace may require a few air cycle-times before a fairly uniform concentration can be assumed [40, 41]. In order to understand the potential infection risk associated with close proximity, we can make a very rough estimate of the exposure in the vicinity of a mask-wearing cougher. The cough momentum will be disrupted by the mask, but the aerosols will exit the mask on essentially all sides [42, 25, 16] resulting in a cloud around the cougher that will then either move with local air currents or rise with the cougher's body plume [43]. We assume that the large droplets are trapped in the mask and the small droplets settle out around the cougher, while aerosols are dispersed into the air around the cougher. The infective dose at a distance d for spherical dispersion is

$$D_{\text{inf}}^{\text{CE}} = \frac{\rho_0 V_c}{N_{\text{inf}}} \frac{3}{4\pi d^3} r_b t. \quad (18)$$

For a typical cough this leads to

$$D_{\text{inf}}^{\text{CE}} \simeq 0.04 \left(\frac{1 \text{ m}}{d} \right)^3 \frac{t}{10 \text{ sec}} \quad (19)$$

which suggests that an infection risk is still present at short distances, even if the cougher is wearing a mask [44]. This analysis is clearly oversimplified, as details of the cough, the mask, and local air currents will prevent isotropic dispersion.

4 Mitigation Measures

The calculations in the previous section allow a variety of possible mitigation measures. This section briefly describes some means of reducing the probability of aerosol infection.

4.1 Masks and Respirators

Masks and respirators can be used to reduce the probability of infection, both for the wearer and for the people they interact with. Standard surgical masks provide some level of protection [45, 46], but they are far less effective filters than N95 respirators (a factor of 2–10 for surgical masks, and 8–80 for N95s) [47]. N95 masks have been demonstrated to be effective in preventing COVID-19 in a hospital setting [48], but benefiting from them requires proper fit and user compliance [49, 50, 51, 52, 53], which suggests that widespread use is likely to be ineffective [54, 55].

Arguably more important than the mask one wears are the masks worn by the people one interacts with, both directly (e.g., in conversation) and indirectly (e.g., by sharing a common space) [56]. Droplets produced while breathing, talking and coughing can be significantly reduced by mask usage [57, 58, 16], but much of the air expelled while coughing and sneezing goes around the mask [42, 25].

4.2 HVAC and Portable Air Filters

Increasing air exchange rates in an HVAC system, and avoiding recirculation can both be used to reduce aerosol concentrations indoors. Consistent use of local ventilation (e.g., bathroom fans) can also help to avoid infection. In buildings and spaces where these measures are not available or not sufficient, local air filters (a.k.a., air purifiers) can be used to increase the introduction of clean air into the space. Small stand-alone units which filter $10 \text{ m}^3/\text{min}$ or more are readily available and could help to increase safety in elevators, bathrooms and small offices.

As noted in sections 3.2 and 5, the particle size of interest is greater than $1 \mu\text{m}$, so special filtering technology is *not* required [39]. Care should be taken, however, when changing filters both in stand-alone units and HVAC systems as they may contain significant viral load. Stand-alone units should be disabled for at least 3 days before changing the filter to allow time for viral infectivity to decay [35]. Building HVAC filters should be changed with proper personal protective equipment, as recommended by the CDC [39].

4.3 Clean Rooms and HEPA Filters

Many laboratory spaces are outfitted with HEPA filters to provide clean air for laboratory operations. Clean rooms offer a clear advantage over other spaces as they are designed to provide air which is free of small particles.

If the HEPA filters are part of a recirculating air cycle, Eqn. 12 can be used with $f_{\text{sys}} \gtrsim 0.999$, allowing for essentially indefinite exposure times. It should be noted, however, that HEPA filters require lower air-speeds than those offered by typical HVAC systems and as such are not a “drop in replacement” option.

Laboratories that use portable clean rooms in large spaces can be treated in a similar manner, using Eqn. 11 for the air inside the clean room and Eqn. 8 to represent the clean air supplied to the laboratory space outside of the portable clean room.

Any clean room environment is likely to provide sufficient air flow to make aerosol infection very unlikely in the well-mixed approximation used in the equations suggested above. This will leave close encounters, as described in section 3.4, as the dominant infection path. If there are multiple occupants in a clean room they should avoid standing close to each other, or being “down wind” of each other [59].

4.4 UV-C Lighting

Illumination of the air-space in patient rooms with ultraviolet light has been shown to dramatically reduce viral longevity in aerosol form, and thereby prevent infection [15, 60]. This could be used to decrease τ_a in spaces where increasing air circulation (i.e., reducing τ_{room}) is impractical. Care is required to avoid exposing occupants to UV-C radiation, which is required for viral deactivation but can

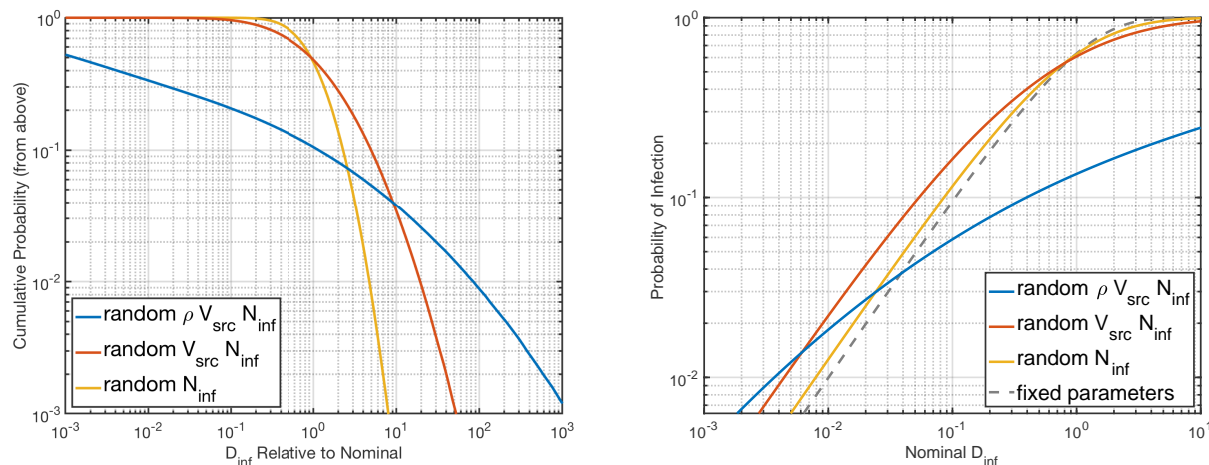


Figure 1: **Left:** Probability that the actual infective dose in any given encounter exceeds the nominal dose by some factor (i.e., $D_{inf} > CD_{inf}^0$ as a function of the dose ratio C). The three curves allow some or all transmission parameters to vary randomly (see section 5 and appendix B). For instance, allowing all parameters to vary (blue trace) the probability of having $D_{inf} > D_{inf}^0$ is about 10%, $D_{inf} > 10 D_{inf}^0$ will happen in about 4% of cases, while 1% of cases will have $D_{inf} \gtrsim 100 D_{inf}^0$. Choosing the nominal viral load ρ_0 equal to the median value of $1/nL$ (rather than the 90th percentile as we have) would shift the blue curve to the right by a factor of 1000. **Right:** Probability of infection as a function of nominal infective dose D_{inf}^0 . The three solid curves relate P_{inf} to D_{inf}^0 allowing some or all transmission parameters to vary randomly (as in the left figure), while the dashed curve (grey) shows P_{inf} with fixed parameters as in Eqn. 1. Allowing all parameters to vary, the probability of infection for a nominal dose of 1 is 14%. A factor of 10 lower D_{inf}^0 reduces the probability of infection to 6%, and $D_{inf}^0 \lesssim 3 \times 10^{-3}$ is required to reduce the probability of infection below 1%. Choosing the nominal viral load ρ_0 equal of $1/nL$ rather than $1000/nL$ would reduce D_{inf}^0 by 1000 relative to our calculations in section 3 and shift the blue curve to the left by 1000, such that P_{inf} would remain unchanged. The “random $V_{src} N_{inf}$ ” curve is used in appendix A to evaluate the probability of infection in cases where ρ is assumed to be high.

be harmful to the eyes and skin. This could be done geometrically (e.g., only illuminate spaces above 2.5 m), or actively with motion sensors.

5 Model Parameters

The parameters used in this model are presented in Table 1. All of these parameters vary between individuals and events, and as such the values given here are intended to serve as a means of making rough estimates which can guide decision making. This section describes the provenance of these values and their variability.

The volume of saliva produced in a variety of activities is used to understand the emission of virions into the environment (known as “viral shedding”). The values given here are for “typical” individuals and behaviors, and actual values for any given person or event may vary by an order of magnitude [16, 33, 34, 61, 62]. Droplets produced while speaking, for instance, depend on speech loudness; speaking loudly, yelling or singing can produce an order of magnitude more saliva than speaking normally [63]. We are careful to avoid quantifying saliva production in terms of the number of droplets produced, since the large droplet-

nuclei are relatively small in number but carry most of the virions (i.e., slope of the number distribution is too shallow to compensate the fact that volume goes with diameter cubed) [33]. For a viral load of $1000/nL$, a $1 \mu m$ droplet nucleus, for example, has only a 1% probability of containing a single virion [63].

The volume of air exchanged with the environment while breathing (“Breathing rate” r_b) scales roughly with the weight of an individual and may vary by a factor of a few. As such, this is a relatively well determined parameter and we use only the nominal value in our calculations. The assumption is that an individual’s tidal volume is about $0.5 L$, and the breathing cycle has a 3 s period [37].

The infectivity decay time of SARS-CoV-2 in aerosol form has been measured and found to be at least a few hours [35, 36]. Note that we are using the $1/e$ decay time, not the half-life, for ease of computation. A separate decay time-scale is set by the settling time of the larger aerosol particles which contain the majority of the viral load. Using the “continuous fallout” model presented in [11] and the data presented in [63] we estimate this as $\tau_{fall} \simeq u_{settle} V_{room} / A_{room} \sim 1$ hour for a room with a 3 m ceiling. In most cases, both the infectivity decay time and the settling decay time are long enough that they be-

come irrelevant as the ventilation system is responsible for removing most of the aerosols.

The viral load in saliva, ρ , has been seen to vary by more than 8 orders of magnitude in individuals that test positive for COVID-19 [30, 31]. Roughly 90% of individuals tested have a viral load less than $\rho_0 = 1000/\text{nL}$, while 1% may have a viral load above $3 \times 10^4/\text{nL}$ [31]. Viral load is seen to decline after symptom onset, so the pre-symptomatic viral load may be on the high-end of the distribution [30, 32, 64]. We use $\rho_0 = 1000/\text{nL}$ for our nominal dose calculations because it results in a rough estimate of the probability of infection relative to the full distribution for probabilities of a few percent (i.e., the “random $\rho V_{\text{src}} N_{\text{inf}}$ ” curve is close to the other curves around $D_{\text{inf}} \sim 0.03$ in Fig. 1, right). This choice does not impact the final probability of infection shown in Fig. 1, since a different choice of ρ_0 would shift the “random $\rho V_{\text{src}} N_{\text{inf}}$ ” curve to compensate.

The respiratory infectivity, N_{inf} , is not well known for SARS-CoV-2, but it has been measured for SARS [27], other coronaviruses, and influenza [23]. Variation by an order of magnitude among individuals is observed both for coronaviruses and influenza. The number we use, $N_{\text{inf}} = 1000$, is intended to be “typical” for coronaviruses and represents roughly 100 “plaque forming units” (PFU) each of which is roughly 10 virions [27].

This analysis is done under the assumption that all workers are wearing some form of face covering (“mask”), however, since effective filtration is difficult to achieve, we assign no protective value to the wearer for mask use. See section 4.1 for more information about masks.

6 Risk Assessment

Many alternatives can be expressed in terms of ratio of the resulting infective dose (e.g., doubling the airflow in a room, or comparing the dose in an office with that of a stairwell), such that the exact parameter values used are not important. However, for computations that require an absolute evaluation of risk, we compute the probability of infection given the distribution of $D_{\text{inf}} \propto \rho V_{\text{src}}/N_{\text{inf}}$, with $V_{\text{src}} = 1 \text{ nL}$, which is common to all dose calculations (see Fig. 1).

The “random $\rho V_{\text{src}} N_{\text{inf}}$ ” curve in Fig. 1 (right) can be used to estimate the probability of a contagious individual causing infection in the people they interact with, given a “nominal infective dose” D_{inf}^0 computed with the parameters in Table 1. For instance, if a brief encounter event delivers $D_{\text{inf}}^0 \sim 10^{-3}$, then the probability of infection is $P_{\text{inf}} \sim 0.4\%$. The infection probability is fairly large despite the tiny *nominal* infective dose because of the non-negligible probability of the contagious individual having a very high value of ρ and thereby delivering $D_{\text{inf}} \gg D_{\text{inf}}^0$ (see Fig. 1, left).

The infective dose can be summed over multiple encounters to compute the probability of infection in at least one

susceptible individual. For example, if a contagious individual delivers a dose of $D_{\text{inf}}^0 \sim 10^{-3}$ and they do it 10 times to each of 10 susceptible individuals (their “cohort”), then the total infective dose is $D_{\text{inf}}^0 \sim 0.1$ and probability of transmission to at least one person is $P_{\text{trn}} \sim 6\%$. (again using Fig. 1, right).

Note that the final probability of transmission is *not* the sum of the infection probabilities for each susceptible individual because of the correlation between these probabilities (i.e., the viral load of the contagious individual is common to all events). It is exactly this correlation that leads to large outbreaks: if a highly-contagious individual delivers an infective dose $D_{\text{inf}}^0 \sim 2$ to many individuals they will each have a 80% probability of being infected (“random N_{inf} ” curve in Fig. 1, right).

The above example shows that limiting the number of potential transmission events between members of a cohort, and limiting the size of a cohort, are both important to minimizing P_{trn} . If only 4 encounters are allowed per person, and the contagious individual only encounters 2 people, then $D_{\text{inf}}^0 \lesssim 0.05$ per encounter gives a total $D_{\text{inf}}^0 \lesssim 0.4$ and a 10% probability of transmission.

The distribution of virions via a building’s HVAC system, discussed in section 3.2, should also be considered as it represents an interaction with a possibly unintended cohort (occupants of rooms connected via the HVAC system). In terms of the previous examples, this behaves as a single encounter with a large group of susceptible individuals.

An acceptable workplace infection risk could be chosen such that an infected employee has only a small chance of infecting another employee during their pre-symptomatic contagious period. The objective is to ensure that the contribution to the epidemic’s R_0 value from the workplace is small, meaning that an employee is more likely to be infected in other activities (i.e., at home). The second example described above (with $P_{\text{trn}} = 0.1$) would contribute 0.1 to R_0 , meaning that for a decaying epidemic with $0.5 < R_0 < 1$, an employee would be 5 to 10 times more likely to be infected away from work than at work.

7 Conclusions

The new world of COVID-19 is here and we will all have to learn to live in it. Understanding the dangers of this new world and how to navigate them will be necessary as we come out of our houses and return to our workplaces.

The calculations presented here suggest that keeping the risk of infection low in the workplace will require both mitigation techniques which lower the viral dose encountered by susceptible individuals, and small interaction groups to avoid large outbreaks should a highly-contagious individual come to work (i.e., a pre-symptomatic “super-spreader”).

Some broad guidelines which we draw from the above analysis are:

1. Aerosol build-up in closed spaces should be treated with care. Avoiding infection requires good ventilation and/or short exposure times. Generally, office spaces should not be occupied by more than one person (see Eqn. 7).
2. Small or poorly ventilated common spaces where many people spend time (i.e., bathrooms and elevators) are of particular concern. At least 4 ventilation cycle times should be allowed to pass between occupants (see Eqns. 13 and 16). Consider increasing airflow and adding local air filters to increase the rate of introduction of clean air.
3. Recirculation in HVAC systems should be avoided if possible. High quality filters (e.g., MERV 12) should be used in recirculating HVAC systems and office use should be kept to a minimum, to avoid transmission through the air circulation (see Eqn. 10).
4. Mask use is critical when a minimum of 2 m interpersonal distance cannot be maintained, but is not sufficient to prevent infection since most masks provide limited filtration of aerosols. Distances less than 1 m remain more dangerous than larger distances due to leakage of aerosols around the mask of a contagious individual (see Eqn. 19).

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A Outbreaks

Documented outbreaks offer a means for checking the plausibility of our aerosol transmission model. In this section we select a few outbreaks where transmission via aerosols appears likely (e.g., distances were too large for droplets, or the pattern of infection followed the airflow).

Information about the outbreaks is, however, limited, and the viral load of the contagious individual is not known, so these comparisons only provide weak constraints on the model parameters. In particular, reported outbreaks are likely to involve unusually contagious individuals (a.k.a. “super-spreaders”), with $\rho_0 \gtrsim 1000/\text{nL}$ (roughly 10% of cases), so we use that value in these computations. Since this fixes the value of ρ , we use the “random $V_{\text{src}}N_{\text{inf}}$ ” curve in Fig. 1 to compute the probability of infection.

A.1 Guangzhou Restaurant

[65] documents a COVID-19 outbreak associated with air flow in a restaurant (Guangzhou, China). The space defined by the wall mounted AC unit is 16 m^2 . Assuming a modest air cycle-time of 30 minutes ($r_{\text{room}} \sim 2\text{ m}^3/\text{min}$), a somewhat talkative contagious individual ($r_{\text{src}} \sim 4\text{ nL}/\text{min}$), and a 30 minute overlap between dinners at different tables, the other dinners were exposed to $D_{\text{inf}} \sim 0.6$. In fact, roughly half of the other dinners contracted COVID-19.

A.2 Hunan Coach

[66] (SCMP article) documents an outbreak on a long distance coach (Hunan, China). This 45 person coach should have $r_{\text{room}} \simeq 8\text{ m}^3/\text{min}$ [10], and we will estimate the ride duration as 2 hours. The contagious individual did not interact with others, so we assign a source rate of $r_{\text{src}} = r_b = 1\text{ nL}/\text{min}$. The resulting infective dose of $D_{\text{inf}} \sim 0.15$ is in reasonable agreement with the fact that 8 of the 45 passengers were infected. The infections were somewhat localized, possibly due to incomplete mixing of the air in the bus.

This outbreak also resulted in the infection of a passenger who boarded 30 minutes after the contagious passenger had disembarked. This could have been due to surface contamination, but then one must wonder why no passengers on later voyages were infected (since fomites last for days) [35]. More likely it indicates that a few air cycle-times passed while the bus was stopped resulting in an order of magnitude reduction in infective dose, and continued during the voyage thereby clearing the air. (Outbreaks on vehicles were common in China, possibly due to poor ventilation [10].)

A.3 Seattle Choir

[67, 68] Of 60 singers 87% infected after singing together for 2.5 hours in a church the size of a volleyball court ($\sim 400 \text{ m}^3$). An air cycle time of 40 minutes gives $r_{\text{room}} \sim 10 \text{ m}^3/\text{min}$, and singing can be approximated by $r_{\text{src}} \sim 30 \text{ nL}/\text{min}$. The 150 minute exposure yields an infective dose of $D_{\text{inf}} \sim 4.5$. Sadly, this makes the probability of infection quite high. Even more sadly, this is not the only choir outbreak [69, 70]. (Like choir practice, aerobic dance classes [71], and call centers [72], are ideal environments for SARS-CoV-2 transmission.)

A.4 Diamond Princess

[73] concludes from the outbreak on the Diamond Princess cruise ship that long-range airborne transmission is unlikely since SARS-CoV-2 did not pass through the ship's air conditioning system. Two Okinawa taxi-drivers were, however, infected by passengers during a shore visit. This outbreak is different from the others in that the contagious individual which initiated the outbreak was not involved in the taxi-driver infections. At the time of the shore visit in Okinawa there were roughly 25 pre-symptomatic infected passengers on the Diamond Princess at least a few of whom disembarked.

If we assume that the taxi ride lasted 10 minutes (the port in Naha is close to the main attractions), that the contagious passenger spent the ride talking to another passenger (or coughed once), and that the taxi had the fan on low ($r_{\text{room}} \sim 1 \text{ m}^3/\text{min}$), the driver was exposed to an infective dose of $D_{\text{inf}} \sim 1$ according to Eqn. 7. Using the "random $\rho V_{\text{src}} N_{\text{inf}}$ " curve in Fig. 1 since these were secondary infections, we find that the probability of transmission was about 15% for each contagious passenger who disembarked. This probability is relatively insensitive to our assumptions and would only change by a factor of 2 if the dose changed by an order of magnitude (in either direction). If both infections in Naha were caused by the same contagious individual (one on the ride in, one on the ride out) it would be appropriate to use the "random $V_{\text{src}} N_{\text{inf}}$ " probability of 60%.

Notably, there were no other COVID-19 infections linked to the Diamond Princess' stop in Okinawa, indicating that the closed environment and long exposure time of the taxi was likely a key ingredient for transmission. That is, changing the air-flow rate in the above calculation to $r_{\text{room}} \gtrsim 100 \text{ m}^3/\text{min}$ to represent shops and more open spaces would reduce the probability of infection to less than 2%. And, while surface transmission could explain transmission to taxi drivers, it does not explain the absence of transmission to shop keepers or other people with whom the passengers of the Diamond Princess interacted.

A.5 Hospital Air Sampling

There was no outbreak in the Nebraska Medical Center, but air sampling in and around COVID-19 patient rooms [74] offers a further cross-check of the calculations presented here. Approximately 3 viral RNA copies per liter of air sampled were found in a patient's room (and in the hallway outside the patient's room after the door was opened). These rooms have $\tau_{\text{room}} \simeq 8 \text{ min}$ and $V_{\text{room}} \simeq 30 \text{ m}^3$, indicating an airflow rate of $r_{\text{room}} \sim 4 \text{ m}^3/\text{min}$ [40]. Equation 5 indicates a steady-state concentration of $\rho_{\text{A}}^{\text{SS}} \simeq 0.25 \text{ /L}$. This suggests that either this patient had a very high viral load ($\rho \sim 10^{10} \text{ /mL}$, which is in the top 3%), or that they were occasionally talking or coughing and had $\rho \sim \rho_0$.

Air sampling data is also available from hospitals and public areas in China. [75] reports quantitative viral deposition rate of roughly 2 /m^2 per minute in a patient's room. The area sampled was 3 m from the patient's bed, so too far for most droplets [11]. If we assume that this is due to slow settling of the larger droplet-nuclei with characteristic a speed of $u_{\text{settle}} \sim 0.1 \text{ m}/\text{min}$ (see section 5 and [11, 63]), this implies a aerosol concentration of roughly 20 /m^3 . (Oddly, air samplers in patient rooms did not detect concentrations above their detection threshold, but the patient's bathroom and other areas in the hospital had concentrations near 20 /m^3).

A concentration of 20 /m^3 is more than a factor of 100 lower than that reported in Nebraska [74], but still implies ρ above the median of the distribution function shown in Fig. 2. If we assume an air flow rate in the room of $r_{\text{room}} \sim 4 \text{ m}^3/\text{min}$ and viral shedding dominated by breathing (i.e., $V_{\text{src}} \sim V_b$), for instance, ρ is roughly $10^8 \text{ /mL} = \rho_0/10$.

B Parameter Probability Distributions

As described in section 5 several parameters used in this work vary significantly between individuals and events. This appendix describe the details of the probability density functions use for ρ , V_{src} and N_{inf} .

Figure 2 shows the distribution for ρ . The estimated distribution used in this work is derived from [31] with the low end of the distribution pushed up somewhat to account for the downward trend in viral load after symptom onset. The log-normal distribution centered at 10^6 /mL with $\sigma = 10^2$ is estimated from the linear fit in figure 2 in [32]. These

distributions both have a median viral load of 10^6 /mL and roughly 10% of cases above 10^9 /mL. We have computed P_{inf} as in Fig. 1 for both distributions and they give quantitatively similar, and qualitatively identical, results.

To account for individual variability, we use a log-normal distribution for N_{inf} which is centered around 1000 and has a width of a factor of 2. This makes the 95% confidence interval 250–4000 which is in reasonable agreement with [27] and [23].

Similarly, an order of magnitude variation among individuals and events is also expected in in droplet and aerosol production [61, 63]. For this we also use a log-normal distribution centered around the V_{src} values given in Table 1 with a width of a factor of 3.

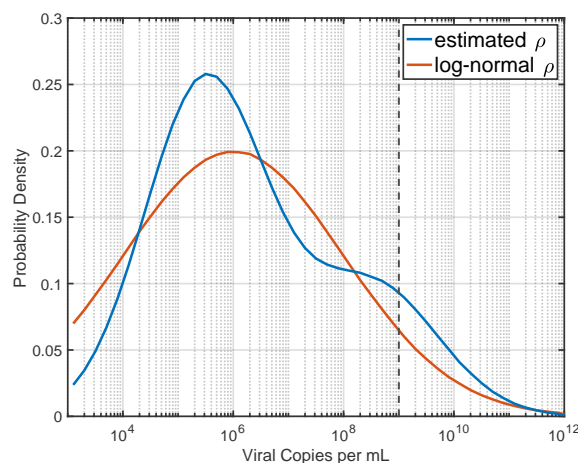


Figure 2: Probability density functions for ρ . The estimated distribution used in this work is shown along with a log-normal distribution centered at 10^6 /mL. The vertical line at 10^9 /mL indicates our “nominal ρ_0 ” of 1000 /nL. 10% of the estimated ρ distribution lies above this line and 7% of the log-normal distribution lies above it.

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