Necessity of ventilation for mitigating virus transmission quantified simply

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To mitigate the SARS-CoV-2 pandemic, officials have employed social distancing and stay-at-home measures. Less attention has focused on ventilation. Effective distancing practices for open spaces may be ineffective for poorly ventilated spaces, both of which are commonly filled with turbulent air. While turbulence initially reduces the risk of infection near a virion-source, it eventually increases the exposure risk for all occupants in a space without ventilation. Here we estimate the time-scale for virions injected into a room of turbulent air to infect an occupant, distinguishing cases of low vs. high initial virion mass loads and virion-destroying vs. virion-reflecting walls. An open window typifies ventilation and we show that its minimum area needed to ensure safety depends only on the ratio of total viral load to threshold load for infection. Our minimalist estimates complement more detailed approaches and present opportunities for generalization.

The SARS-CoV-2 virus, first reported in 2019 \[1, 2\] has since spread to at least 213 countries and territories leading to an unprecedented global pandemic \[3\]. The lack of therapeutics and vaccines has led public health officials to employ non-pharmaceutical interventions focused on physical distancing measures and masks, but comparatively much less on ventilation. With plans for the resumption of visits to offices, bars, restaurants, salons and universities, where people are expected to be in close proximity in for prolonged periods of time, ventilation and HVAC filtering are safety precautions that must be prioritized \[4, 5\].

Airborne viral particles like SARS-CoV-2, which cause Covid-19, get trapped on moisture droplets that carry the virions \[6\]. Interior air is commonly, if not unavoidably, turbulent. This keeps droplets airborne much longer than their free-fall time \[7\]. Since virions follow droplets, basic principles of turbulent diffusion and transport or aerosols \[8–15\] become directly relevant to guiding HVAC issues and the efficacy of masks \[16\]. Forced central air/heating in HVAC systems without sufficient replenishment of fresh air exacerbates the danger of airborne virions. The rapid spread of droplets in room of turbulent air is exemplified by spraying a scented aerosol and measuring how quickly a person on the other side of the room can detect it. The benefits of physical distancing are diminished by prolonged exposure to viral filled turbulent air in a closed room because virions are transported throughout by turbulent diffusion of the host droplets. Without a tightly sealed mask and proper eye protection, accumulated indoor-exposure is likely.

Virions can also be transported by HVAC systems between rooms. People staying home may be exposed to the virus in poorly ventilated apartment buildings with forced circulating air. Evidence for such non-local transport of viruses has been found in restaurants in China \[17, 18\], a call-center in South Korea \[19\], and a choir-setting in Washington state \[20\]. The study of airborne disease transmission and ventilation has a empirical history \[23–25\].

Much about the transmission modes of SARS-CoV-2 remains unknown, including the viral load required to cause infection \[21\]. Empirical answers are unlikely without detailed experiments that may take a year or more. It is therefore particular important to identify key principles that inform policy choices to reduce the risk of transmission \[4\]. As such, here we use basic concepts of turbulent transport to provide minimalist order-of-magnitude estimates.

Specifically, we estimate the time scale for an individual to be infected in an enclosed space of arbitrary size, subjected to an injection of virions. We distinguish between virion-absorbing vs. virion-reflecting walls and cases in which the injected mass of virions is sufficient vs. insufficient to infect a person over one diffusion crossing time from the virial injector to the room boundary. We consider the role of an open window as a proxy for ventilation, and estimate the typical cross-section needed for safety. Since the threshold viral load for infection and the different loads produced by talking, breathing and other modes \[26, 27\] are unknown, we present our results in terms of a dimensionless quantity—the mass required to infect a single individual. Our results demonstrate the importance of ventilation and filtering, and the approach provides a user-friendly, complement to more detailed computational efforts \[7, 17, 22\].

To begin, we assume that a viral load bound to a droplet of mass $M$ is injected at the center of a room of radius $R$, and that the room has steady turbulent air of local eddy air speed $v_l$ and eddy scale $l \ll R$. We de-
fine $t_c$, as the time scale for a single person to get infected and $M_e$ as the viral load needed to infect that person. These two quantities are related by

$$t_c = M_e/M_h,$$  

where $M_h$ is the rate of mass encountering a human face. We make the reasonable assumption that the threshold virus load for infection is small compared to the total viral content in the room. In Table I we list the variables for the calculations that follow.

We first consider the case of a room with no ventilation, for which the injected viral mass is above the threshold for infection. Then if the viral mass were spread uniformly over the room, the mass flux encountered by a single passing of the diffusion front across a facial cross-section $A_h = h^2$, where $h$ is the typical radius of a face, would be sufficient for infection. The total mass of virions in the room is $M = 4\pi R^3 \rho/3$, and that encountering a facial cross-section is $M_e = 4\pi h^3 \rho/3$. Therefore we have

$$M/M_e \geq 1.56 \times 10^4 \left( \frac{R}{5m} \right)^3 \left( \frac{h}{0.25m} \right)^{-3},$$  

where we use a room of $25m^2$ and a typical face-size of $0.2m^2$ as reference scales. In this limit, the time scale to infect any individual is bounded by the diffusion time from the injection point to the room boundary. Assuming isotropic turbulence, the eddy diffusivity is $\nu_T = v_l/3$.

The critical time for infection is therefore

$$t_c \leq R^2/\nu_T = 3t_l \left( \frac{R}{5m} \right)^2 \leq 1.67 \left( \frac{t_l}{0.75m} \right) \left( \frac{R}{5m} \right)^2 \leq 2 \text{ min},$$  

Here $t_l = l/v_l$ is the eddy turnover time and its scaling of 0.75s was estimated using an eddy scale of $l = 0.75m$ corresponding to injection by a room fan at a flow speed 1m/s.

We now consider the case for which the initial injection of viral mass is small enough such that

$$M/M_e \ll 1.56 \times 10^4 \left( \frac{R}{5m} \right)^3 \left( \frac{h}{0.25m} \right)^{-3},$$  

meaning that the introduced viral mass is insufficient to infect anyone after one diffusion front crossing of the room. Assuming again that there is no ventilation, we must next consider two limiting cases of interest: (i) completely absorbing walls (say if the walls are infused with anti-viral material such as copper fibers) and (ii) completely reflecting walls. In the former case, virions are removed upon contact with the wall and no one in the room is infected.

For the latter case, we assume that virion-carrying droplets remain airborne and the inequality, Eq. (4) allows us to assume they are well-mixed throughout the space, before any one individual can be infected. Given that the accumulated mass of virions on a single person is

$$\dot{M}_h \approx \rho v_{\text{diff}} A_h,$$

where

$$\rho = \frac{3M}{4\pi R^3}$$

and $v_{\text{diff}}$ is the diffusion speed, which is the turbulent diffusion coefficient $\nu_T$ divided by the net displacement from the source to point of measurement. Given that we are in the well-mixed regime, the relevant displacement is just the eddy scale since any new virion-mass accumulates via neighboring eddies. Therefore,

$$v_{\text{diff}} = \frac{1}{3} v_l.$$  

Combining Eqs. (5), (6), and (7) into Eq. (1) yields, for the critical infection time,

$$t_c = 4\pi \times t_l \left( \frac{M_e}{M} \right) \left( \frac{R}{5m} \right)^2 \left( \frac{h}{0.25m} \right)^{-1}.$$  

To facilitate numerical estimates, we scale as before, and write equation (8) as

$$t_c = 13.11 \left( \frac{t_l}{0.75m} \right) \left( \frac{M/M_e}{50} \right)^{-1} \left( \frac{R}{5m} \right)^3 \left( \frac{h}{0.25m} \right)^{-2} \text{ min},$$

with reference to a room of viral mass equivalent to infecting 50 people. Note, that while $M_e$ is unknown, in all cases it enters as as fraction of the input viral mass. In the upper panel of Fig. 1 we plot Eq. (9) as a function of the room radius $R$ and injected load $N = M/M_e$. By definition, to prevent infection an occupant must spend a duration $t \leq t_c$ in the room.

Next we focus on the effect of ventilation, the simplest example of which is a window of open area $W << R^2$, that is allowed to exchange both interior and exterior air.
Assuming pressure and temperature equilibrium everywhere, from turbulent mixing, air that diffuses through the open window gets replaced with an equivalent total mass of virus-free air. With these assumptions, viral mass escapes from the room at a rate of

$$\dot{M}_W = \rho v_{\text{diff}} W.$$  

From the time of viral-mass injection, it takes a single diffusion time from the source to the window for exterior air to interact with the viral air. This has no effect for the case when the viral mass exceeds the critical threshold for a single infection as in Eq. (2). Nor does it have any bearing for absorbing walls, where the viral load is absorbed over one diffusion front crossing.

The ventilation is however influential for the reflecting wall case that led to Eq. (9), which now requires modification. For this case, the relevant time scales are $t >> R^2/\nu T$. The viral mass lost in some time $t$ can be estimated as $M_L(t) = M_W t$. Dividing this by $M$ and using equations (6) and (10) gives the fractional loss of viral mass over this time scale

$$M_L(t)/M \simeq \frac{1}{4\pi} \left( \frac{v_l t}{R^2} \right) \left( \frac{W}{R^2} \right).$$  

Safety would be achieved when $M_L(t)/M \simeq 1$. Solving Eq. (11) for the associated $t = t_{\text{saf}}$ gives,

$$t_{\text{saf}} \simeq 4\pi R^3 \left( \frac{W}{v_l} \right).$$  

Next we set Eq. (11) equal to Eq. (12) to get the minimum open window area $W_c$ ensuring $t_{\text{saf}} < t_c$, so that enough virions are removed before anyone gets infected. This gives

$$W_c \geq 2 \left( \frac{h}{0.2m} \right)^2 \left( \frac{M/M_c}{35} \right) m^2.$$  

This is independent of the room size because the same viral mass in a larger room reduces the viral density. Therefore the flux of mass incident on a face, as well as on the surface of a window is reduced by the same factor. In the lower panel of Fig. 1b we show $t_c/t_{\text{saf}}$ as a function of $W$ and $N$. The intersection of the curved surface with the plane is equation (13).

Given that we have assumed $\rho$ to be uniform by the diffusion action of turbulence, the ratio of mass flux through a window to that encountering a person’s face is approximately $W/h^2$. Increasing this ratio enhances the probability for viral mass encountering the window. Our simple example shows that the strategy for safety is to increase this ratio such that the leftover viral density in the room is insufficient to produce a critical infectious load for individuals during their period of stay in that enclosed space.

To summarize: turbulence is hard to avoid in interior spaces. When the viral mass injected into a room is sufficiently high, a person can be infected over one turbulent diffusion time, as per equation (3). More likely are poorly ventilated spaces where occupants are only exposed to a threshold load over longer times. For reflecting walls and no open windows, the time scale for infection is given by Eq. (9) and illustrated in the upper panel of Fig. 1. Spending more time than this in such an unventilated room would lead to infection, independent of physical distancing.

To highlight the role of ventilation, we estimated the minimum size of an open window needed to mitigate infection for a one-time viral load for the case that would otherwise cause infection on the time scale of equation (9). The critical window size is given by Eq. (15) and depends only just one unknown, $M_c$. The lower panel of Fig. 1 exemplifies the effect, showing that a window area $\geq 2m^2$ is enough to prevent occupants being infected for a viral load that could potentially infect 50 people. Open windows are extremely helpful.

FIG. 1. Critical time for infection and condition for safety. Upper panel: the time $t_c$ for a non-ventilated room with reflecting walls according to Eq. (9) plotted as a function of the number of people that can be infected by the injected load and room radius $R$. Infection is prevented by spending a time $t \leq t_c$. Lower panel: mitigation by introducing ventilation via an open window of area $W$. The planar surface is the critical surface $t_c/t_{\text{saf}} = 1$ above which the time scale for infection $t_c$ exceeds the time scale $t_{\text{saf}}$ for which enough virions have diffused through the window. Points on the curved surface above the plane indicate safe occupancy duration. The line of intersection between the surfaces shows the window area needed as a function of the viral mass injected into the room according to Eq. (13).
Retrofitting HVAC systems with UVC (ultra violet c) or other anti-viral filters can also reduce exposure, particularly from air passing between rooms in building complexes. Interior walls also provide a useful surface area. If imbued with anti-viral materials and constructed to mitigate boundary layer effects, rooms could potentially be safe after one turbulent diffusion time, subsequent to viral load injection. For any viral load satisfying equation [4], infection would be prevented. Such measures are also effective for other airborne illnesses such as the flu and offer the economic benefits of reducing sick days, even in non-pandemic circumstances.

Precision is not required for our key results and message, but its pursuit warrants detailed numerical models with generalizations. These include turbulent and droplet size spectra; differing airborne survival times; droplet-eddy coupling times as a function of droplet and eddy sizes and humidity; repeated injection of viral loads from different room-locations varying mass load levels \(26, 27\); time-dependent viral mass injection; temperature gradients \(28\); room geometry, time-dependent, anisotropic inhomogeneous turbulence; and wall boundary layer effects \(22\).

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