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Sensitivity of airborne transmission of enveloped viruses to seasonal variation in indoor relative humidity

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\textbf{ABSTRACT}

In temperate climates, the peak in infection rates of enveloped viruses during the winter is likely heightened by seasonal variation in relative humidity within indoor spaces. While these seasonal trends are established in influenza and human coronaviruses, the mechanisms driving this seasonality remain poorly understood. Relative humidity impacts the evaporation rate and equilibrium size of airborne particles, which in turn may impact particle removal rates and virion viability. However, the relative importance of these two processes is not known. Here we use the Quadrature-based model of Respiratory Aerosol and Droplets to explore whether the seasonal variation in enveloped viruses is driven by differences in particle removal rates or by differences in virion inactivation rates. Through a large ensemble of simulations, we found that dry indoor conditions typical of winter lead to slower virion inactivation than humid indoor conditions typical of summer; in poorly ventilated spaces, this reduction in inactivation rates increases the airborne concentration of active virions, but this effect was important to virion exposure only when the susceptible person was farther than 2 m downwind of the infectious person. On the other hand, the impact of relative humidity on particle settling velocity did not significantly affect the removal or travel distance of virus-laden particles, suggesting that relative humidity is more likely to affect seasonal transmission via inactivation rates than via particle removal.

\textbf{1. Introduction}

Late autumn to early spring is often referred to not as the winter season, but as the flu season, reflecting the annual resurgence of influenza viruses it brings with it. The high winter infection rates documented for influenza\textsuperscript{[1]} are also observed in other airborne viruses sharing its enveloped structure and upper respiratory infection site\textsuperscript{[2,3]}. One likely cause of this trend is the seasonal variation of conditions within the indoor environment\textsuperscript{[4]}. While outdoor temperature and humidity levels vary dramatically throughout the year, humans spend around 90\% of their time inside\textsuperscript{[5,6]} where temperatures remain stable but relative humidity (RH) displays consistent summer highs and winter lows\textsuperscript{[4,7]}. Several mechanisms pertinent to airborne transmission of viruses are sensitive to this variability in RH, such as faster inactivation of enveloped virions at extreme RH levels\textsuperscript{[3,4]} and shorter travel distance of mid-sized particles at high RH levels\textsuperscript{[8]}. However, it is unclear which mechanism - RH-mediated differences in droplet removal or RH-mediated differences in virion inactivation - drives the seasonality of enveloped viruses.

In the context of familiar airborne diseases like the flu or the winter common colds (caused by human coronaviruses (HCOVs) 229E, HKU1, NL63, and OC43)\textsuperscript{[9,10]}, understanding the mechanisms driving this relationship between RH and the seasonality of infection rates is a retrospective question; hundreds of years of evidence have clearly established their seasonality\textsuperscript{[11]}, and current research helps clarify why, not if, seasonal cycles have occurred. In the case of emerging viruses like SARS-CoV-2, however, the question is preemptive: will transmission vary seasonally in the future? The first year of an outbreak cannot answer this question\textsuperscript{[12,13]}, but establishing whether novel viruses are likely to follow seasonal trends is critical to preparing adequate mitigation strategies\textsuperscript{[11,14,15]}.

Determining the impacts of RH on airborne transmission is vital to progressing our understanding of the seasonal dynamics of any enveloped virus. However, quantifying those impacts requires a model holistic enough to account for the series of processes governing airborne transmission and efficient enough to span the tremendous variability in...
relevant conditions. Most models complex enough to provide a detailed representation of short-range and long-range airborne transmission (e.g. [16,17]) are too computationally expensive to represent the large uncertainty in indoor conditions, particle properties, and physiological parameters. To obtain a mechanistic understanding of the impact of RH on airborne transmission while also accounting for the large uncertainties in model parameters, we performed thousands of simulations using the new Quadrature-based model of Respiratory Aerosol and Droplets (QuaRAD) [18]. We use the transmission of SARS-CoV-2 as an example due to its current relevance; however, conclusions are also relevant to other prominent winter viruses that share its enveloped virion structure [19] and ability to transmit through the airborne route [20].

In this paper, we first describe QuaRAD (Section 2), emphasizing specific details about the interaction of RH with evaporation (Section 2.1) and viral inactivation (Section 2.2). We then examine the impacts of typical summer and winter RH levels on exposure in a single baseline scenario (Section 3.1) and across a large ensemble of scenarios (Section 3.2). Finally, we discuss the insights gained on the seasonality of airborne enveloped viruses and the utility of different mitigation strategies (Section 4).

2. Model description

We assessed the impact of typical indoor RH levels on virion fate and transport using QuaRAD, a broad overview of which is given below. The full model description, including specific parameter distributions, can be found in [18]. The model approximates the size distribution of and viral load within expired particles using numerical quadrature. Such quadrature-based moment methods have been shown to accurately represent integrals over aerosol distributions with only a small number of particles [21,22].

QuaRAD models the evolution, dispersion, and removal of aerosol particles and droplets as they are transported through the expiratory jet of an infectious person and through the broader air motions of the indoor space. The simulated spatiotemporal variation in the size distribution of infectious particles is used to estimate the number of virions inhaled by a susceptible person. The particle size distribution is represented with three superimposed lognormal modes, based on the measurements of speech emissions detailed in [23]. We estimate the viral loads associated with each particle using these size distribution measurements in combination with Gesundheit II and quantitative RT-PCR measurements of influenza virion emission on fine and coarse particles [24,25]. We assume that influenza is a reasonable model for viral emissions of SARS-CoV-2 [26]. In QuaRAD, the size distribution of respiratory particles and its evolution is represented using only six weighted particles, which provide an accurate quadrature approximation of integrals over the distribution. This quadrature representation allows for simulation of large ensembles of scenarios that span the uncertainty in model parameters.

For each quadrature point, we simulate the evaporation (Section 2.1) and the subsequent virion inactivation (Section 2.2). Particle dispersion within the expiratory jet of an infectious person is modeled using a Gaussian puff model [27] in combination with a buoyant turbulent jet model [28], following the approach of [8]. All simulations include particle removal through gravitational settling and deposition to the walls, floor and ceiling, such that removal rates vary with the size distribution. We also represent particle removal through ventilation, assuming that air is exchanged with outdoor air and is not recirculated through the indoor space. The total number of virions inhaled by a susceptible person is computed as the sum over the quadrature points. Input parameters are sampled from the realistic probability distributions detailed in [18]; the baseline scenario discussed in Section 3.1 was simulated using the median of the probability density functions from which the ensemble inputs were sampled.

In this study, we analyzed the continuous stream of aerosol particles and droplets expelled when an infectious person sneezes and the subsequent exposure of a susceptible individual, which we quantified as the number of virions inhaled per second $N_{\text{inhale}}$. $N_{\text{inhale}}$ was computed using the concentration of virions present at the mouth of the susceptible person, their average number of breaths per minute, and the average volume of air inhaled per breath. We quantify the difference in active virion inhalation between RH cases as described in Section 2.3.

2.1. Evaporation

Upon expiration, aerosol particles and droplets evaporate to an equilibrium size that depends on their composition and the environmental conditions. Following the approach of [8,29] and implemented as detailed in [18], we model evaporation by solving a coupled pair of ordinary differential equations representing the mass and heat transfer:

$$\frac{dm_p}{dt} = 2\pi p D_p M_v D_{sw} C_v S_h (\frac{p - p_{v,\infty}}{p - p_{v,\infty}})$$

Eq. (1) describes the temporal evolution of the particle mass $m_p$ as a function of time $t$, ambient pressure $p$, particle diameter $D_p$, molecular weight of water $M_v$, diffusivity of water in air $D_{sw}$, correction factor $C_v$, Sherwood number $S_h$, gas constant $R$, surface vapor pressure $p_{v,\infty}$, and ambient vapor pressure $p_{v,\infty}$. Eq. (2) describes the evolution of the particle temperature $T_p$ as a function of the particle’s specific heat $C_p$, the air thermal conductivity $k_a$, the background temperature $T_\infty$, the Nusselt number $Nu$, the latent heat of vaporization $L_v$, and Eq. (1). These parameters are provided in [18]. The model represents convection-induced evaporation enhancements. We assume that particles are spherical and contain 1%–9% non-water aerosol components by volume, following the measurements of [30]. The vapor pressure over an aqueous droplet is computed from the $\kappa$-Köhler model [31].

Seasonal changes in RH modify $p_{v,\infty}$, such that droplets evaporate more slowly with increasing RH, as shown in Fig. 1. RH is the ratio between $p_{v,\infty}$ and the saturation vapor pressure, which depends on temperature. Although QuaRAD represents the variation in temperature and RH within the turbulent jet of an infectious person, we found that results were insensitive to this variation. Therefore, we assume that expired particles encounter the background temperature and humidity as soon as they are expired.

2.2. Viral inactivation

Like other enveloped viruses [4], SARS-CoV-2 virions exhibit exponential decay [32] with comparable rates on surfaces and while airborne.
The inactivation of a virion eliminates its ability to infect a cell and is analogous to the death of an organism. The trends of inactivation due to temperature and RH are established, though the exact inactivation rates remain poorly constrained; since our focus is on the indoor environment, we do not consider the accelerated inactivation rates caused by UV light [13]. As with influenza [4,34] and other coronaviruses [35,36], SARS-CoV-2 decays more rapidly at high temperatures than low temperatures and shows a U-shaped dependence on RH, as seen in Fig. 2. At room temperature, for example, virion half-lives are longer than 6 hours for RH < 45% or RH > 85% but are around 2.5 h for RH ≈ 65% [32].

Several mechanisms may cause the temperature dependence of viral inactivation, including thermal denaturation [4,32,37] and decreased lipid ordering in viral envelopes at high temperatures [4,38]. The RH dependence of viral inactivation is likely caused by RH’s influence over solute concentrations in infectious particles. Solutes are thought to function as reactants in virion inactivation, and the U-shaped humidity dependence is thus theorized to result from two ways in which the interior solute concentrations are reduced [32,39]. At low RH levels, evaporation causes solutes in expired particles to effloresce, separating from the aqueous solution as solids on the particle surface. If the RH within the indoor space is above a threshold, known as the efflorescence relative humidity (ERH), solutes within the aqueous particle will remain in the solution. In this regime, the solute concentration decreases with increasing RH because, as long as the RH remains above the ERH, will remain more dilute the less water is removed. For this reason, inactivation rates are fastest just above the ERH, when solutes are in solution and are also highly concentrated.

In this model, we used the measured distributions of SARS-CoV-2 half-lives in a cell culture medium at three temperatures (283.15 K, 295.15 K, 300.15 K) and RH levels (40%, 65%, 85%) from [32] to estimate the distribution of viral decay rates k given in Fig. 2. Though we recognize that ERH varies with particle size and composition, we followed [32] in assuming an ERH of 45% across particles. These values were chosen due to the explicit variation in environmental conditions but are comparable to those obtained in [19,33,40]. Given the focus of this paper on seasonal indoor environments, we analyzed the measurements taken at T = 295.15 K, corresponding to an average indoor temperature of 22°C, and at RH = 40% or RH = 65%.

2.3. Quantifying changes in exposure with RH

For simulations that isolate individual mechanisms, we quantify the relative difference between the virion inhalation rates, \( N_{inhale} \), at two RH levels:

\[
\Delta_r = \frac{N_{inhale,RH=40\%} - N_{inhale,RH=65\%}}{N_{inhale,RH=65\%}}
\]  

Positive values of the relative difference \( \Delta_r \) indicate an increase in the inhalation rate of active virions with a decrease in RH from 65% to 40%. To identify which mechanisms drive the seasonal variation in the simulations, we compare \( \Delta_r \) for simulations that include different combinations of processes. The selected RH levels match the conditions under which [32] measured virion half-lives and approximately correspond to the median indoor RH of 62% in the summer and 42% in the winter measured by [41]. Susceptible individuals are assumed to inhale at a rate of 14 breaths per minute and a volume of 4.69 \times 10^{-4} m^3 per breath in the base case and at 12 to 20 breaths per minute and 3.75 \times 10^{-4} to 6.25 \times 10^{-4} m^3 per breath across the ensemble [18].

3. Results

In this section, we quantify how two key mechanisms influence the sensitivity of virion inhalation rate to RH, which is assessed as a function of downwind distance from an infectious individual. Before describing the effect of RH on airborne transmission across an ensemble of simulations in Section 3.2, we first show how RH impacts key processes in an example scenario in Section 3.1.

3.1. Sensitivity of virion exposure to RH in a single scenario

In the baseline scenario, we found that the sensitivity of \( N_{inhale} \) to RH depends on the distance between the infectious and the susceptible person (Fig. 3). When directly downwind of an infectious person, \( N_{inhale} \) is insensitive to RH, whereas we find a 10% increase in \( N_{inhale} \) with a decrease in RH from 65% to 40% at distances greater than 2 m (solid line in Fig. 3B). When inactivation is neglected and only seasonal differences in particle removal rates are simulated, \( \Delta_r \approx 0 \) both near and far from the infectious person (dotted line in Fig. 3B).

If a susceptible person is within the expiratory jet of an infectious person, nearly all virions they inhale would have been expelled from an

![Fig. 2. Measured SARS-CoV-2 virion half-lives under different conditions adapted from [32]. We focused on the half-lives of 6.43 h at RH = 40% and 2.41 h at RH = 65% measured at room temperature (shown in purple), corresponding to inactivation rates of 0.108 h^{-1} and 0.288 h^{-1}, respectively. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.]

![Fig. 3. The (A) virion inhalation rate \( N_{inhale} \) and (B) relative difference between RH levels \( \Delta_r \) computed with Eq. (3) in the baseline scenario with respect to downwind distance after 1.5 h. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)]
infectious person just seconds earlier and, therefore, would not be influenced by changes in virion inactivation that occur over much longer timescales. On the other hand, far from an infectious person, a large portion of virions may be contained in particles that were expelled hours earlier. For this reason, $N_{\text{inhale}}$ far from an infectious person depends strongly on the RH-dependent inactivation rates ($\Delta_r \approx 0.1$), whereas near-field exposure to $N_{\text{inhale}}$ is insensitive to changes in inactivation rates ($\Delta_r \approx 0$).

3.2. Factors governing sensitivity of virion exposure to RH across ensemble

To quantify the mechanisms driving the sensitivity to RH across the wide range of conditions expected in indoor spaces, we performed an ensemble of 1000 scenarios using the distributions in input parameters described in [18]. As in the base case, $N_{\text{inhale}}$ was insensitive to RH when directly downwind of an infectious person. Beyond distances of approximately 2 m, $N_{\text{inhale}}$ was higher at a typical winter RH of 40% than at a typical summer RH of 65%. The mean $\Delta_r$ (Fig. 4) follows the same trend as the base case. We again found that the sensitivity of $N_{\text{inhale}}$ to RH was driven by differences in virion inactivation and not by differences in particle removal through gravitational settling and deposition.

Since RH significantly affects transmission only when the susceptible person is far from the infectious person, we explored the sensitivity of $N_{\text{inhale}}$ to conditions controlling far-field virion concentrations: the duration of the encounter and the ventilation rate of the room (expressed here in air changes per hour, or ACH). If the infectious person and the susceptible person are in a poorly ventilated space, the virion concentrations throughout the room increase over time. Under these conditions, virion exposure is highly sensitive to inactivation rate, as shown by the high $\Delta_r$ values at low ventilation rates in Fig. 5. Virion concentrations also increase as the duration of the encounter increases. Consequently, longer interactions correspond with higher exposure and a higher $\Delta_r$ (comparison between $\Delta_r$ in a 1-hour interaction (green) and in a 4-hour interaction (purple) in Fig. 5).

We also found that high ventilation rates reduce the dependence of $\Delta_r$ on the length of the encounter. For example, when the ventilation rate is less than 1.0 ACH, the median $\Delta_r$ after four hours is nearly twice the median $\Delta_r$ after one hour, but when the ventilation rate is greater than 3.0 ACH, the median $\Delta_r$ values after four hours and after one hour are approximately equal.

4. Discussion

4.1. Comparison between mechanisms

Our process analysis revealed that the greater virion exposure at RH $= 40\%$ than at RH $= 65\%$ was caused by differences in virion inactivation rate and not by RH-induced changes in particle removal rates through gravitational settling and deposition. We also found that pathogen transport over short distances was insensitive to RH. In indoor spaces, SARS-CoV-2 virions have long half-lives ($h = 6.4 \pm 0.03$ hours at RH $= 40\%$ and $h = 2.4 \pm 0.04$ hours at RH $= 65\%$), so nearly all virions in freshly expired particles are active, regardless of the background RH. Whereas virion exposure directly downwind of an infectious person is driven by virions in freshly expired particles, virion exposure far from an infectious person may be strongly influenced by particles that remain suspended for hours.

Whereas changes in inactivation rates with RH led to seasonal differences in virion survival within respiratory particles, removal rates and travel distances of the respiratory particles themselves were insensitive to seasonal changes in RH. RH does affect the evaporation time-scale and equilibrium size of expired particles (see Fig. 1), but the impact of those differences on travel distance is limited by the expired size distribution. Close to the infectious individual, most expired particles are small enough to travel with the flow of the respiratory jet at their initial diameter, during evaporation, and at their equilibrium diameter; most of the other expired particles are large enough to quickly settle out of the respiratory jet at their initial diameter, during evaporation, and at their equilibrium diameter. Far from the infectious individual, the equilibrium sizes of most particles at RH $= 40\%$ and RH $= 65\%$ are too similar to cause major differences in particle travel or residence time. The plume does contain mid-sized particles whose spread and settling velocity are sensitive to RH, but these particles make up only a tiny proportion of those expired during speech and consequently have very little effect on $N_{\text{inhale}}$. While this finding does not contradict earlier findings of RH sensitivities in the spread of specific particles, it does show that those differences have very little impact on transmission risk when integrating over a population of polydisperse particles.

4.2. Sensitivity to timescale and ventilation rate

Analysis across the ensemble revealed that at 4 m of distance, RH has a much greater impact on virion exposure over the course of a long encounter than over the course of a short one. Far-field concentrations increase over time, such that inhaling air in the far-field after only a few
minutes of emissions results in a much lower $N_{inhale}$ than after an hour of emissions; near-field concentrations do not show this time dependence because, in close proximity, $N_{inhale}$ is controlled by the freshly expired virions in the expiratory jet instead of the virions building up over time throughout the room. Because longer timescales result in greater virion concentrations than shorter timescales and the decay rate of far-field virion concentrations depend on RH, longer timescales thus allow the decay rate to have a greater impact on $N_{inhale}$, resulting in an elevated $\Delta_i$.

Far-field concentrations are also affected by the ventilation rate of the room. Ventilation removes particles from the air, so when the ACH is low, far-field particle concentrations build up over time. When particles linger for a long time, the difference in inactivation rate between RH scenarios results in a high $\Delta_i$. On the other hand, when the ACH is high, far-field particle concentrations are reduced more quickly. The impact of RH on exposure thus decreases as the ACH increases, resulting in a small $\Delta_i$ in well-ventilated spaces. We thus find that the impact of RH on far-field exposure over long timescales can be meaningfully reduced by increasing the ventilation rate.

4.3. Study limitations

While we found that typical winter and summer indoor RH levels indeed impact virion exposure, it is very likely that other factors not accounted for in this model are also in play. For example, immune response capabilities of susceptible individuals may be impaired by seasonal deficiencies in vitamin D [42] or RH-mediated reductions in mucociliary clearance [43], each of which may increase the probability of winter infections. Frequency of exposure may also be determined by seasonal differences in human behavior, including the school calendar and the higher frequency of indoor social events in colder seasons [44, 45]. We also assume that air is exchanged with outdoor air, so we neglect the impact of RH on pathogen transport through filters. We also note that QuaRAD accounts solely for seasonal differences in airborne transmission; while this is likely the dominant transmission route of SARS-CoV-2 [46] and a prominent one for other discussed viruses, differences in droplet or fomite transmission may independently impact seasonality.

5. Conclusion

In this paper, we showed that a low RH increases airborne virion exposure by slowing the virion inactivation rate, not by increasing the travel distance or residence time of respiratory particles. In poorly ventilated spaces and encounters lasting four hours, we found that lowering the RH from 65% to 40% led to a 20% increase in the median virion exposure by slowing the virion inactivation rate, not by increasing the ventilation rate.

The observed sensitivity of virion exposure to RH is reduced with higher ventilation and shorter exposure times, suggesting high ventilation and shorter interactions are critical to limiting winter transmission risk even when following safe social distancing guidelines. In settings where short exposure times are impossible or where multiple groups of people transition between the same room, such as classrooms, hospitals, factories, or prisons, employing other protections like effective mask wearing and high ventilation are critical to limiting transmission risks.

Credit author statement

LF was responsible for conceptualization, methodology, software, and funding acquisition. AR and LF were responsible for investigation, visualization, and writing, review and editing. AR was responsible for writing the original draft.

Data availability

The QuaRAD source code, input files, and processing script are available for download at: https://github.com/lfierce2/QuaRAD/. Simulation ensembles were created using latin hypercube sampling with pyDOE: https://pythonhosted.org/pyDOE/.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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