# Modeling the impact of indoor relative humidity on the airborne transmission of several respiratory viruses risk using a modified Wells-Riley model

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**Abstract.** There is good evidence supporting the airborne transmission of many respiratory viruses (measles, influenza A, human rhinovirus and the novel SARS-CoV-2). Relative humidity (RH) is an important factor in understanding airborne transmission as it may impact both airborne survival, inactivation by biological decay, and the gravitational settling of the virus-laden droplets. This study aimed to estimate and compare the impact of indoor relative humidity on the airborne infection risk caused by these viruses using a novel modified version of the Wells-Riley model. To gain insights into the mechanisms by which relative humidity might impact airborne transmission infection risk, we modeled the size distribution and dynamics of airborne viruses emitted from a speaking person in a typical residential setting over a relative humidity (RH) range of 20–80% at a temperature of 20-21 °C. Besides the size transformation of virus-containing droplets due to evaporation and then removal by gravitational settling, the modified model also considers the removal mechanism by ventilation. The direction and magnitude of RH impact depended on the respiratory virus. Measles showed a highly significant RH impact that was as strong as the ventilation impact, as the infection risk was roughly the same at RH of 13.5 % and 6 ACH compared to a higher RH of 70 % and 0.5 ACH. For other viruses, ventilation dominated over RH. In the case of SARS-CoV-2, a very high RH of 83.5% was needed to reduce the infection risk. For rhinovirus, however, the high RH of 80% increased the infection risk. Within the acceptable range of RH of 20-50% indoors, our modeling showed that RH had practically no impact for SARS-CoV-2 and rhinovirus, while the upper RH significantly reduced the infection risk of influenza A at the lowest ventilation rate of 0.5 ACH. This relative impact of RH on infection risk became very weak at higher ventilation rates of 2-6 ACH independently of the virus types (except measles). In conclusion, we showed that in well-ventilated rooms, RH range of 20-50% did not affect the airborne risk of influenza A, SARS-CoV-2, and rhinovirus.

**Keywords.** Virus airborne transmission, Ventilation, Relative humidity, Indoor environment, Wells-Riley model
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## 1. Introduction

There is good evidence supporting the airborne transmission of many enveloped respiratory viruses (measles [1], influenza A (H1N1) [2], MERS-CoV [3], and the novel SARS-CoV-2 [4]) and certain non-enveloped viruses (human rhinovirus [5]). Airborne infections pose a particular threat to susceptible individuals whenever they are placed together with an infected person in confined spaces [6]. It is therefore important to understand the risks posed by infectious individuals in indoor environments so that interventions can be developed to minimize the spread of airborne infection. In this context, predictive mathematical models and risk assessments can be a fundamental tool for understanding and planning effective infection control strategies in indoor environments. Wells-Riley model is the classic model to quantitatively assess airborne infection risk; it is based on the seminal work of Wells [7] and Riley et al. [8]. The Wells-Riley model has extensively been used to evaluate the airborne infection risk of respiratory diseases [9]. The original Wells-Riley model has been extended to account for three sink or removal mechanisms – ventilation, gravitational settling, and biological decay of the airborne pathogen [10]. However, the removal terms of gravitational settling and biological decay in this model can only be calculated for one specific environmental condition being RH. This is a limitation of the model, as RH may affect the airborne transmission of respiratory viruses via both the deposition loss and airborne decay of infectious droplets [11]. To gain insight into the mechanisms by which relative humidity might impact airborne transmission infection risk, we modeled the size distribution and dynamics of airborne viruses emitted from a speaking person in a typical residential setting over a relative humidity (RH) range of 20–80% and at a temperature of 20-21 °C. Our model advances a mechanistic understanding of the aerosol transmission route, and results complement recent studies on the relationship between humidity and respiratory disease infectivity. It is an extension of the work published earlier investing only the impact of RH on the risk of infection with SARS-CoV-2 [12].

## 2. Methodology

A schematic representation of the theoretical model assessing the impact of RH on the quanta emission rate of SARS-CoV-2 for infection risk assessment is shown in Fig 1.

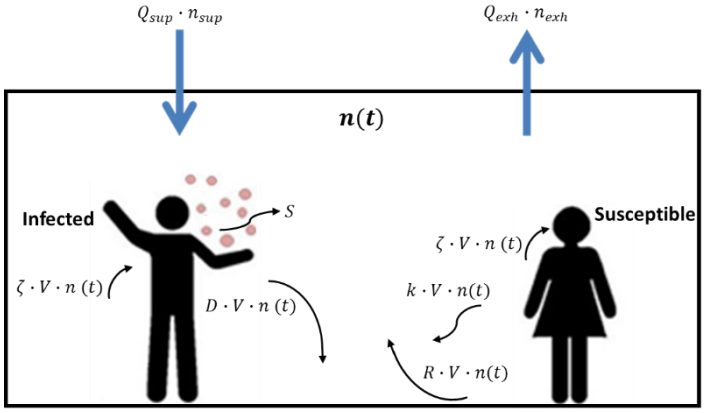


**Fig 1.** Schematic representation of a simple indoor air mass-balance model in a completely mixed environment including a source term S and removal mechanisms by ventilation, inactivation by biological decay *k*, deposition by gravitational settling *D,* resuspension *R,* and respiratory absorption ζ.

The mass balance model for a completely mixed indoor mechanically ventilated room model can be represented by the following differential equation representing a single-zone model:

$$V \cdot \frac{dn(t)}{dt} = S + Q_{sup} \cdot n_{sup}(t) - Q_{exh} \cdot n_{exh}(t) - k \cdot n(t) \cdot V - D \cdot V \cdot n(t) - R \cdot V \cdot n(t) - 2 \cdot \zeta \cdot V \cdot n(t) \quad (1)$$

To solve equation (1) in the form of a first-order differential equation $\frac{dn(t)}{dt} + n(t) \cdot a = b$, it may be rewritten as follows:

$$\frac{dn(t)}{dt} + n(t) \cdot \left(\frac{Q}{V} + D + k + 2 \cdot \zeta\right) = \frac{S}{V} \quad (2)$$

The unique solution of quanta concentration in an indoor environment with complete mixing ventilation at time t, $n(t)$ is:

$$n(t) = n_0 \cdot e^{-\left(\frac{Q}{V}+D+k+2\cdot\zeta\right)\cdot t} + \frac{S}{V} \cdot \left\{ \frac{1}{\frac{Q}{V}+D+k+2\cdot\zeta} - \frac{1}{\frac{Q}{V}+D+k+2\cdot\zeta} \cdot e^{-\left(\frac{Q}{V}+D+k+2\cdot\zeta\right)\cdot t} \right\} \quad (3)$$

where $n_0$ is the initial quanta concentration ($\frac{quanta}{m^3}$) at time t = 0.

To perform calculations with (9) to predict indoor concentrations of quanta at time *t*, appropriate expressions for the source term *S*, deposition rate *D*, inactivation rate *k*, and absorption rate ζ must first be known. Regardless of the type of respiratory virus considered, the deposition rate *D* and absorption rate ζ will be calculated in the same manner. The detailed description of calculating the

impact of RH on deposition rate $D$ can be found in [12], while the calculation of the absorption rate ζ can be found in [13].

The pollutant source emission rate $S$ is defined as the quanta emission rate of respiratory virus generated by infected persons in the room divided into $n$ bins and can be defined by: $S = I \cdot c_v \cdot c_i \cdot IR \cdot \sum_{i=1}(N_i \cdot V_i)$ (4)

$I$ – number of infected persons, -

$N_i$ - droplet number concentration in the i[th] bin, $\frac{particles}{cm^3}$

$V_i$ – the mean volume of a single droplet (mL) in the i[th] bin.

$$V_i(D) = \frac{\pi \cdot (D_{max}^4 - D_{min}^4)}{24 \cdot (D_{max} - D_{min})} \quad (5)$$

where $D_{max}$ and $D_{min}$ denote the bin's lower and upper diameter values [14].
$i$– size bin of the droplet distribution.

The size distribution for talking is determined experimentally by the works of Morawska et al. [15] for droplet aerosols $\leq 2\ \mu m$ and Chao et al. [16] for respiratory droplets $\geq 2\ \mu m$: both studies measured the size distribution of droplets for talking/voice counting at a distance of 10 mm from the participant's mouth opening.

$IR$ , $\frac{m^3}{h}$– inhalation rate. The inhalation rates for resting and standing averaged between males and females are equal to 0.49 and 0.54 $\frac{m^3}{h}$, respectively [17].

$c_v$ – viral load in the sputum , $\frac{RNA}{ml}$ or $\frac{\text{TCID50}}{ml}$

$c_i$ – conversion factor is defined as the ratio between one infectious quantum and the infectious dose expressed in viral RNA copies (quanta/RNA) or tissue culture infectious dose ($TCID_{50}$/ml). The mean values for $c_i$ and $c_v$ are given in Tab 1. as derived in a recent study by Mikszewski et al. [18]

**Tab 1.** Mean viral load $c_v$ and conversion factor $c_i$ values for different respiratory viruses [18] carried by droplets $\leq 5\mu m$ in a dehydrated state

| Respiratory virus | $Log_{10}$ $c_v$ | Conversion factor $c_i$ | S (quanta/h) |
|---|---|---|---|
| Measles | 3.5 $TCID_{50}$/ml | 1.0 quanta/ $TCID_{50}$ | $14.79 \cdot 10^{-3}$ |
| Influenzia | 6.7 RNA/ml | $7.1 \cdot 10^{-6}$ quanta/RNA | $0.166 \cdot 10^{-3}$ |
| Rhinovirus | 3.6 $TCID_{50}$/ml | 0.053 quanta/ $TCID_{50}$ | $0.99 \cdot 10^{-3}$ |
| SARS-CoV-2 | 5.6 RNA/ml | $1.4 \cdot 10^{-3}$ quanta/RNA | $2.6 \cdot 10^{-3}$ |

To characterize the impact of relative humidity on the inactivation rate, experimental data on the survival time of the respiratory viruses in aerosols were used for measured values of $k$ ($min^{-1}$) at RH = 20%, 50% and 80 % at T = 20–24 °C, as shown in Tab 2.

**Tab 2.** Mean inactivation rates $k$ $[min^{-1}]$ for the considered respiratory viruses at different RH values

| Respiratory virus | Relative humidity (RH %) | Temperature (°C) | Mean inactivation rate k $[min^{-1}]$ |
|---|---|---|---|
| Influenza A [19] | 21 | 20-24 | $8.68 \cdot 10^{-4}$ |
| | 50.5 | | $9.65 \cdot 10^{-3}$ |
| | 81 | | 0.013 |
| Measles [20] | 13.5 | 20-21 | 0.010 |
| | 69 | | 0.112 |
| Rhinovirus [21] | 30 | 20±1 | 0.066 |
| | 50 | | 0.066 |
| | 80 | | $1.21 \cdot 10^{-3}$ |
| SARS-CoV-2 [12] | 20 | 20-25 | 0.0103 |
| | 53 | | 0.0101 |
| | 83.5 | | 0.0314 |

To determine the probability of infection ($P$, %) as a function of the exposure time ($t$) of susceptible people, the quanta concentration was integrated over time through the Wells–Riley equation as follows:

$$P = \left(1 - e^{-IR \int_0^T n(t)dt}\right) \quad (\%) \quad (6)$$

## 3. Results

We show preliminary results for a 40 $m^2$ x 3 m room with two occupants, one infected person, and one susceptible person that is distanced by at least 1 m; only droplets less than 5 microns in size before evaporation (dehydrated state) are considered for all cases. Fig 2. shows the impact of RH and ventilation rate on the infection risk when the person is infected with influenza A. The trend is clear - the infection risk decreased with higher RH. However, the overall effect of RH on the infection risk must be interpreted as a function of both the ventilation rate and time. The shorter the time interval and higher the ventilation rate, the less RH has an impact on the infection risk. It can be noted that for higher ventilation rates (6 ACH) the relative impact of RH on the infection risk is very weak.

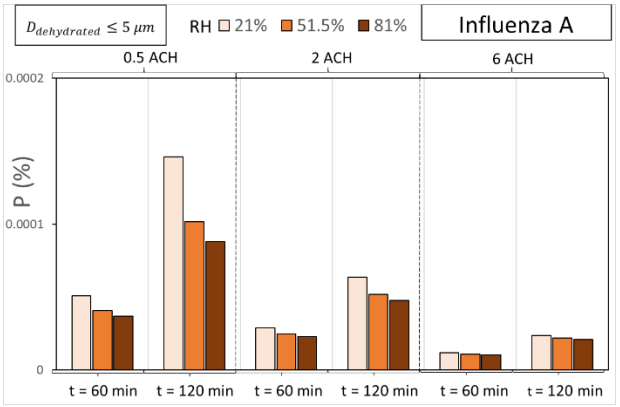


**Fig 2**. Impact of RH and ventilation (ACH = Air Exchange Rates per Hour) on the infection risk probability P (%) when influenza A infected person with a viral load of $c_v = 10^{6.7}$ RNA/ml is speaking continuously for 60 and 120 min.

The impact of RH on infection risk when a person is infected with measles is similar to the case scenario with influenza A as depicted in Fig 3. Very low RH values of 13.5 % significantly increase the infection risk when compared to a higher RH value of 70 %. Although again the relative impact of RH decreases higher ventilation rate the overall impact of RH is still prominent.

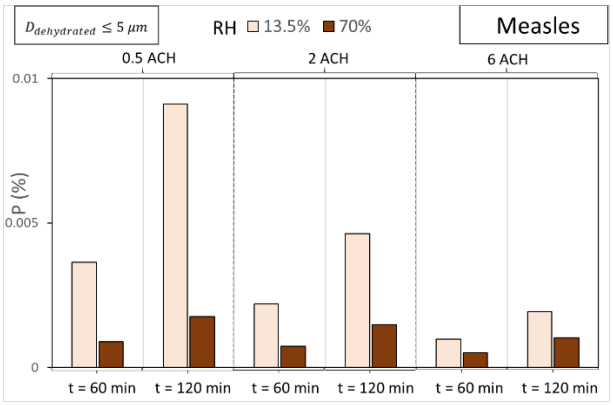


**Fig 3.** Impact of RH and ventilation (ACH) on the infection risk probability P (%) when the measles infected person with a viral load of $c_v = 10^{3.5}$ $TCID_{50}$/ml is speaking continuously for 60 and 120 min

Compared to influenza A and measles, the impact of RH on the infection risk for rhinovirus is inverse - the infection risk significantly rises for a higher relative humidity of 80 %, while it is lower for RH 30% and 50%. The difference between the calculated infection risks at RH = 30% and 50% is almost non-existent.

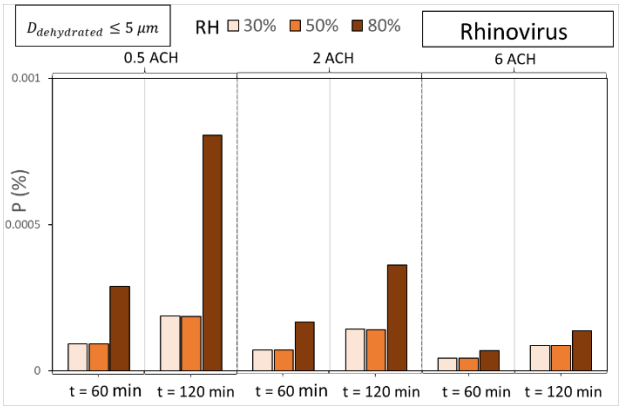


**Fig 4.** Impact of RH and ventilation (ACH) on the infection risk probability P (%) when a rhinovirus infected person with a viral load of $c_v = 10^{3.6}$ $TCID_{50}$/ml is speaking continuously for 60 and 120 min

Lastly, the impact of RH on SARS-CoV-2 infection risk is shown in Figure 5. The infection risk is lower for high RH values of 83.5 %, but there is no significant difference between infection risks for RH values between 20% and 53%. The difference between the infection risk at all three RH values considered becomes almost non-existent at higher ventilation rates (6 ACH).

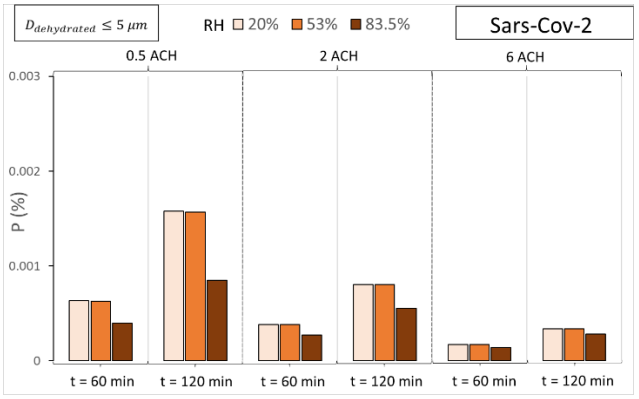


**Fig 5.** Impact of RH and ventilation (ACH) on the infection risk probability P (%) when a SARS-CoV-2 infected person with a viral load of $c_v = 10^{5.6}$ RNA/ml is speaking continuously for 60 and 120 min

## 4. Conclusions

The infection risk for four different respiratory. iruses (influenza A, measles, rhinovirus, and SARS-CoV-2) was estimated using the modified Wells-Riley model at different RH levels and different ventilation rates for a specific indoor scenario when an infected person was continuously talking for 120 min. We considered dehydrated droplets having a size lower than 5 microns. We assumed complete mixing and did not examine the impact of RH on susceptibility. The findings of the study can be summarized in the following key points:

- Four respiratory viruses showed different impacts of RH on infection risk regarding the direction of the impact and the magnitude compared to the ventilation rate impact
- For influenza A and SARS-CoV-2, the infection risk decreased with the highest RH values of 81% and 83.5%, but differences were small in the RH range relevant for indoor spaces. While for influenza A the infection risk was slightly lower at RH=51.5% compared to 21%, for SARS-CoV-2 there was practically no effect in the 20-53 RH range.
- Increasing RH from 20 or 30% to 50% had practically no effect in the case of SARS-CoV-2 and rhinovirus and had a small but significant positive effect in the case of influenza A. This impact of RH was more prominent for lower ventilation rates (0.5 ACH) and became very low at higher ventilation rates (6 ACH).
- For rhinovirus, the infection risk significantly rose for a higher relative humidity of 80 %, however, the difference between the calculated infection risks at RH = 30% and 50% was almost non-existent.
- Measles was the only virus for which RH impact was as strong as the ventilation impact, the infection risk was roughly the same at RH of 13.5 % and 6 ACH compared to a higher RH of 70 % and 0.5 ACH demonstrating a highly significant impact of RH.

In conclusion, we showed that maintaining a higher ventilation rate may have a more beneficial effect on reducing the airborne risk of four different airborne viruses than changing RH. Our model advances a mechanistic understanding of the aerosol transmission route, and results complement recent studies on the relationship between humidity and respiratory disease infectivity. The modified model can be used by public health experts, engineers, and epidemiologists when selecting different measures to reduce the infection risk from various respiratory diseases indoors allowing informed decisions concerning indoor environmental control

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