INTRODUCTION

In order to limit the spread of COVID-19, a first response is to isolate contagious people from others as soon as they are detected. Home quarantine is usually preferred for first-stage symptoms to limit hospital room congestion. As recommended by WHO, when home quarantine is chosen, the person should occupy a well-ventilated single room, or if a single room is not available, maintain a distance of at least 1–2 m from other household members, minimize the use of shared spaces and cutlery, and ensure that shared spaces (such as the kitchen and bathroom) are well ventilated. Other competent authorities ask to avoid direct contact when interacting with contaminated people by arranging groceries to be dropped at the door and to wash hands directly before and after any interaction with others (EU) or the ill person should put the toilet lid down before flushing and wearing a mask, including a non-medical mask, can help protect others (Canada).

All those recommendations aim to limit close contact and fomite exposure that were supposed to play a major role in the transmission of SARS-CoV-2 at the beginning of the pandemic. However, evidence of contamination via air transport of viral aerosol has rapidly emerged thanks to the international Indoor Air Quality (IAQ) Community. The extended short-range aerosol spread of SARS-CoV-2 having occurred in a poorly ventilated and crowded restaurant is one well-known example. Anderson et al. analyzed three sets of evidences: some case reports of transmission for asymptomatic individuals under normal breathing and talking without coughing (so subject to aerosol transport), limited empirical data that have...
recorded aerosolized SARS-CoV-2 particles (remaining suspended in air for hours and subject to spread over distances including outside of rooms and within buildings), a literature on the importance of aerosol transmission of other infectious diseases. They concluded that airborne aerosol transmission of SARS-CoV-2 is significant. This conclusion is also supported by the analysis of Hadei et al., who relate the past experiences and knowledge about the mechanisms of similar viruses such as SARS-CoV suggest that airborne transmission may be involved in the case of SARS-CoV-2. In this way, IAQ experts have alerted the international authorities to recognize that the virus is spread by air and recommend that adequate control measures on the use of ventilation be taken as various studies showed that SARS-CoV-2 is transmitted by respiratory droplets. The different routes of air transmission are multiple: direct transport of droplets exhaled by an infected person to the mouth, nose, or eyes of a person, direct contact of droplets deposited on surfaces and via aerosol containing virus in the air and are then inhaled. It has been demonstrated that the infection risk is especially high indoors and that there are important reasons to suspect that aerosol transmission plays a role in an indoor environment when social distancing and washing hands are respected.

The present paper aims at evaluating the SARS-CoV-2 virus spread in a single-family house when one sick occupant is confined in his bedroom. No other pathways are accounting in the analysis, that is, it is an ideal perfect quarantine where the other residents can only be in contact with the virus transported from the bedroom of the contagious people via intake of the infected air. In a first part, we present the numerical methodology that is employed to evaluate the aerosol concentration within the house, along with the input data and studied configurations. The results are presented and discussed in the consecutive parts in terms of exposure to the contaminated droplets in the whole house and infection relative risk to other residents when confinement actions are employed. In a last part, the effects of virus variants and vaccination are also investigated.

2 | METHODS

2.1 | Building description

The building (Figure 1) is similar to the four main rooms individual house defined in the QUAD-BBC study, that is, walls, windows, ceilings, and floors have the same composition and heating temperature set points and scenarios were also selected according to this study. Heat (lighting, computer, cooking) and moisture (washing dishes, showering, doing laundry, or drying machines) sources are typical of a French residential building. Statistics on ventilation systems installed in houses showed that 35.7% are mechanical systems, 29.2% natural systems and the rest of the house stock with no centralized systems or no system at all. The ventilation system considered for this study is the most commonly installed in French houses from the 1980s, that is, a whole-house pressure-controlled exhaust ventilation. Note that, with the RT2012 building energy standard, a humidity-controlled system is now preferred in new buildings because of its reduced airflow rates during inoccupancy. In whole-house ventilation (either pressure- or humidity-controlled system), outside air enters the house by the main rooms (bedrooms and living room) through inlets usually located above the windows and is extracted in the kitchen, bathroom, and toilet by outlets located on the ceiling. All the system elements, that is, the pressure-controlled inlets and outlets, the exhaust ducts and fan, have been considered according to manufacturer specifications (the characteristic curves of the inlets and fan are presented in Figure 1 for illustration). The two-speed fan allows a high airflow rate of 180 m³/h during cooking activity and a lower one of 105 m³/h. The high airflow rate is activated only 30 min during cooking time (at 12:00 a.m. and 07:30 p.m.). As a consequence, the exhaust fans provide an air change rate of 0.44 and 0.75 vol/h at the low and high settings. The envelope air-tightness is assumed homogeneous in the whole dwelling and corresponds to an envelope permeability index of Q₄₀₀ = 0.9 m³/(h.m²) (n₅₀ = 1.8 ACH). The airflow rate per wall surface area is given in Figure 1 as a function of the pressure gradient between indoor and outdoor. Air infiltration through the ceiling of the rooms located in the second floor is also considered. No other leakages are accounted such as those from the floor or through the internal walls.

2.2 | Simulation procedure

This numerical work was conducted using the coupling process between CONTAM and TRNSYS softwares. Zone temperature and humidity are evaluated by TRNSYS considering the weather, the heating system, and the indoor heat/moisture sources. CONTAM calculates the airflows between rooms (and outdoor) by taking into account the wind pressure on the building envelope through the use of pressure coefficients with wind velocity and direction, the air pathways (envelope airtightness, ventilation inlets, open doors, door undercuts, open windows, etc.), and stack effect. Multizone airflows are looped on TRNSYS inputs to update multizone airflow and calculate back temperatures. Aerosol droplet concentration is
then calculated in each room considering perfectly well-mixed volume. The mass balance of droplets is performed considering interzonal transport by airflow, aerosol source located in Room 3 and decay rates induced by both deposition and virus deactivation. The risk for other residents is then calculated by a dose-response model (see below) that depends on various parameters (virus concentration in droplets, respiratory deposition according to droplet sizes, etc.) and, in particular, the droplet concentration and the time of exposure. Regarding the latter parameter, a typical schedule of an exposed occupant downstairs during the day and in Room 2 at night was considered.

2.3 | Aerosol and virus sources

In order to study the aerosol-like virus spread, it is necessary to address the size distribution of droplets emissions by the contaminated occupant of Room 3. Human expired aerosols result from different mechanisms of production and different zones. Morawska et al. measured the concentration of droplets of different size for various activities: breathing, coughing, and speaking. Table 1 presents an adaptation of this work to considered size bins.

The estimated source of particles, last column of Table 1, is largely based on breathing data. However, in order to bring a correction on the nodal repartition of aerosol droplets, the following scenario has been considered: The contaminated occupant is supposed to be speaking for 200 s every hour, representing only 5.6% of the day, and coughing 80 times per hour. Morawska et al. data are based on an average cough interval of 1.2 s, and therefore, coughing represents approximately 3% of the day in our study.

This scenario aims to represent a resting individual without any severe form of the disease. The effect of speaking and mild coughing on the breathing rate has been neglected, and a global rate of 0.36 m³/h has been used for all activities (breathing, speaking, and coughing). A fever or severe acute respiratory syndrome could increase this breathing rate and, therefore, the droplet source.

The viral load in droplets can be approximated from the recently documented viral load in sputum and saliva \( c_v \) (copies/ml). To et al. measured a viral load in saliva between \( 10^3 \) and \( 10^8 \) copies/ml in the first 10 days after symptom onset. Most patients showed an antibody response after this period and the virus load dropped in most cases. These results are in good agreement with results from the nasopharyngeal area, ranging from \( 10^6 \) to \( 10^{10} \) copies/ml for the 15 first days of symptom duration.

However, droplets are solutions of salts and proteins and a fast equilibrium occurs with ambient humidity and in dwellings this phenomenon usually leads to a reduction of droplet diameters by a factor close to 2 and so the volume is reduced by a factor close to 8 (concentration factor \( \alpha \)). Thus, it can be expected that the viral load per millimeter in droplets is higher than the one in the sputum.
they are originated from. Indeed, in the dataset used to estimate the sources, it has been considered that this equilibrium occurred before the measurements. Therefore, our study already takes into account most of the size change in time of aerosol droplets, from the respiratory system to the indoor air conditions. However, we neglected further modifications due to slight changes of temperature and relative humidity during the lifespan of the aerosol droplets inside the house.

Despite a global decrease in viral load with time, we decided to adopt a constant value of $10^7$ copies/ml for the viral load in aerosol droplets ($\alpha C_v$), for the 2 weeks duration of our study.

In the case the source occupant is wearing a mask, we introduced a factor $M_E = 0.5$ on the source emission rate for all droplet sizes. Because of the multiplicity of mask types, it was not possible to evaluate droplet size-dependent mask efficiency so the same mean value from Davies et al. has been chosen. Indeed, most of the efficiency drop is due to air leakage around the mask which does not depend on the size bins.

### 2.4 Active virus decay rate and surface deposition

One of the elimination processes is due to the inactivation rate ($k$) of the virus outside the human body. A correspondence of The New England Journal of Medicine (2020) estimates the half-life of CoV-19 virus at 1.09 h for a relative humidity of 65% and a temperature of about 21°C. The corresponding decay rate, or inactivation rate, is $k = 0.63 \text{ h}^{-1}$. This value is lower, but of the same order, than the ones observed for the flu. Surface deposition is calculated from deposition velocity ($V_d$) using Lai and Nazaroff" model for smooth surface (Table 2). Both are integrated in the simulation procedure simultaneously with air flow modeling.

### 2.5 Dose-response model

Once the virus source determined, the previously described simulation procedure allows us to assess the concentration of contaminated particles in the building. Nonetheless, other factors are to be considered to measure the dose received by the occupants. In this study, we will consider the possibility to wear masks and the retention factor of droplets in the respiratory system, depending on their size. We therefore chose a dose-response approach.

The quantity of copies inhaled by an occupant, for a time $t$, can be expressed as a dose ($d$) following equation:

$$d = \alpha C_v \cdot IR \cdot M_E \cdot \int_0^t \sum_{i=1}^8 N_i(t) \cdot V_i \cdot RDF_i \, dt \quad (1)$$

where IR is the inhalation rate ($\text{m}^3/\text{h}$) of the occupant (around 0.54 m$^3$/h for a person standing with a moderate activity), $N_i$ (particles/$\text{m}^3$) and $V_i$ (ml) are, respectively, the concentration and the volume of the droplets of the size bin $i$. $RDF$ (no unit) is the respiratory deposition fraction for each size bin, or retention factor of the droplets, and is estimated from Teske et al. ($1 - M_E$) is the efficiency of the mask worn by the exposed occupant. $M_E$ equals 1.0 if no mask is worn and 0.7 otherwise. $M_E = 0.7$ signifies that the mask efficiency in reducing the virus inhalation by the exposed occupant is set to 0.3. Davies et al. reported a filtration efficiency of 0.5 for perfectly fitting masks, but as Jimenez and Peng suggested, this value is very conservative and a value between 0.3 and 0.5 is much more reasonable. As before, the same value for all size bins is considered as it is not only due to filtration efficiency but also air leakage around the mask.

### Table 1: Droplets concentration in breath and estimated emission rate of a contaminated occupant

| Mean diameter of the size bin (µm) | Droplets concentration in breath (#/ml) | Estimated source of particles $S_{part}$ (particles/h) |
|-----------------------------------|----------------------------------------|-----------------------------------------------------|
|                                  | Breathing                               | Coughing                                             |
| 0.34                             | $2.3 \times 10^{-2}$                    | $8.3 \times 10^{-2}$                                 |
| 0.55                             | $3.8 \times 10^{-2}$                    | $2.0 \times 10^{-1}$                                 |
| 0.89                             | $3.5 \times 10^{-2}$                    | $2.3 \times 10^{-1}$                                 |
| 1.44                             | $1.2 \times 10^{-2}$                    | $8.1 \times 10^{-2}$                                 |
| 2.34                             | $3.5 \times 10^{-3}$                    | $3.0 \times 10^{-2}$                                 |
| 3.79                             | $3.1 \times 10^{-3}$                    | $8.5 \times 10^{-3}$                                 |
| 6.16                             | $1.4 \times 10^{-3}$                    | $2.8 \times 10^{-3}$                                 |
| 10.0                             | $3.7 \times 10^{-4}$                    | $1.8 \times 10^{-5}$                                 |

### Table 2: Deposition velocity on floor, ceiling and vertical walls

| Mean diameter of the size bin (µm) | Deposition velocity (cm/s) |
|-----------------------------------|----------------------------|
|                                  | Floor          | Ceiling        | Vertical walls |
| 0.34                             | $5.11 \times 10^{-4}$ | $5.73 \times 10^{-12}$ | $2.79 \times 10^{-5}$ |
| 0.55                             | $1.17 \times 10^{-3}$ | $2.81 \times 10^{-31}$ | $1.84 \times 10^{-5}$ |
| 0.89                             | $2.81 \times 10^{-3}$ | $0$            | $1.26 \times 10^{-5}$ |
| 1.44                             | $6.96 \times 10^{-3}$ | $0$            | $8.72 \times 10^{-5}$ |
| 2.34                             | $1.76 \times 10^{-2}$ | $0$            | $6.16 \times 10^{-4}$ |
| 3.79                             | $4.53 \times 10^{-2}$ | $0$            | $4.41 \times 10^{-4}$ |
| 6.16                             | $1.17 \times 10^{-1}$ | $0$            | $3.20 \times 10^{-6}$ |
| 10.0                             | $3.07 \times 10^{-1}$ | $0$            | $2.35 \times 10^{-6}$ |
Considering the probability of inhaling pathogens distributed in a random manner in the medium and the probability for each inhaled pathogen to survive and initiate the infection, the risk of illness \( R_d \) at a dose \( d \) can be expressed by an exponential model:\(^{28}\):

\[
R_d = 1 - \exp\left(-\frac{d}{\lambda}\right)
\]  

(2)

where the infectivity parameter, \( \lambda \), represents the reciprocal of the probability that a single pathogen will initiate the response.

### 2.6 | Infectivity estimation

The infectivity parameter \( \lambda \), previously introduced, is one of the most challenging to estimate. Most studies are carried on animals and require interspecies extrapolation.

Watanabe et al.\(^{28}\) sought to estimate this parameter for SARS-CoV-1. The only data set reported for humans, in their study, gives an estimate of \( \lambda \) of 19 for HCoV-229E, a coronavirus linked to colds. Based on other data on mice, Watanabe et al.\(^{28}\) concluded on an infectivity value of around \( 4 \times 10^4 \) for SARS-CoV-1. A recent study on ferrets\(^{29}\) was also used by Zhang and Wang\(^{30}\) and led to an estimation of \( 6.4 \times 10^4 \) for the infectivity of SARS-CoV-2 in case of an airborne source.

These data give us a large range estimate of \( \lambda \) from \( 10^1 \) to \( 10^5 \).

The incertitude on the infectivity of SARS-CoV-2 led us to consider another approach, the Wells–Riley model, in order to compare our inputs with epidemiological data with the idea of making a more accurate estimate. This model is based on the concept of quantum to implicitly consider a large scale of parameters, such as the infectivity, infectious source strength, and biological decay of pathogens.\(^{31}\) A quantum is defined as the number of infectious airborne particles required to infect and may be one or more airborne particles.\(^{22}\) This kind of models can, therefore, be used to perform risk assessment without the infectious dose data of the pathogen as it can be calculated epidemiologically from an outbreak case. Despite not being equivalent, under certain hypotheses, it is possible to link the quantum to the infectivity. The goal is, here, only to benchmark our approach to the literature available with Wells–Riley type models. Some of the assumptions made are solely for comparison purpose.

In case of a single source occupant, the risk of illness \( R_{WR} \) of an occupant breathing at rate \( IR \) (m\(^3\)/h) during a time \( t \) can be expressed according to the Wells–Riley Equation (3), under steady-state and well-mixed assumptions:

\[
R_{WR} = 1 - e^{-dRIQ}
\]  

(3)

where \( q \) is the quantum generation rate of the source (copies/h), considered constant, and \( Q \) is the volumetric airflow rate of uncontaminated air in the room (m\(^3\)/h).

Assuming ventilation dilution as the only sink at a rate \( Q \), we can approximate the concentration of virus copies in the air \( C_{\text{copies}} \) (copies/m\(^3\)) as

\[
C_{\text{copies}} = \frac{\sum_{i=1}^{8} S_{\text{copies},i}}{Q} = \alpha c_v \sum_{i=1}^{8} N_i(t)V_i
\]  

(4)

where \( S_{\text{copies},i} \) represents the source of virus copies for a size bin \( i \) (copies/h).

From Equations (2), (3), and (4), given these hypotheses, the quantum generation rate \( q \) can be directly compared to \( S_{\text{copies}} \) following Equation (5):\(^{32}\)

\[
\frac{\sum_{i=1}^{8} \text{RDF}_i S_{\text{copies},i}}{\lambda} \approx q
\]  

(5)

It is then possible to benchmark our approach, detailed in Table 3, with the quantum estimates available in the literature and reported in Table 4. For this comparison, we used the minimum value of the interval for \( \lambda (\lambda = 10) \), that is, the maximum infectivity. Despite the fact the assumptions allowing this comparison are not made in our simulations, it allows us to position our work with epidemiological data that are using a Well–Riley approach.

This benchmark gives us several insights. Indeed, despite the choice of the lowest value of the determined range for \( \lambda \), the estimation of the corresponding quantum generation rate is in good agreement with the ones determined for other similar diseases or SARS-CoV-2. In this study, the source occupant is considered resting with a slow respiration rate. It is therefore consistent to be in the low range of possible values, as the source is proportional to expiration rate in our approach. The quantum equivalent calculated in Table 3 also ignores inactivation rate and deposition rate for the comparison. These phenomena are considered in the following results, and

| TABLE 3 | Comparison of our approach with the Wells-Riley model quantum under strong hypotheses and \( \lambda = 10 \) |
|-----------------|---------------------------------|----------|-----------|
| Mean diameter of the size bin (\( \mu m \)) | Estimated source of virus copies \( S_{\text{copies}} = \alpha c_v S_{\text{part}} V_i \) | RDF\(^{26}\) | RDF \( S_{\text{source}} \) (equivalent quanta/h) |
| 0.34 | 0.21 | 0.13 | 0.003 |
| 0.55 | 1.6 | 0.18 | 0.029 |
| 0.89 | 7.6 | 0.36 | 0.27 |
| 1.44 | 15 | 0.63 | 0.95 |
| 2.34 | 31 | 0.85 | 2.6 |
| 3.79 | 72 | 0.94 | 6.8 |
| 6.16 | 128 | 0.93 | 12 |
| 10.0 | 118 | 0.84 | 9.9 |
| Total of all bins | 374 | – | 32 |
a true equivalence between the approaches would lead to a lower quantum generation rate.

Moreover, for SARS-CoV-2, it has been shown that the highest infectivity is observed for a maximal contribution of the airborne source.\textsuperscript{30} Available literature on infectivity originates from data on animals and could be higher on humans, as shown in a study for HCoV-229E.\textsuperscript{28}

This comparison and observations argument in favor of the choice a high infectivity for our study. Following results are given for $\lambda = 10$.

### 2.7 Studied configurations

Table 5 summarizes the five configurations designed to study the effects of the actions to isolate and decrease the virus concentration of Room 3. Case #0 is the reference case where no action is taken and where the door opening happens similarly for all rooms (from 6:30 a.m. to 10:00 p.m.). This case was selected as a reference as it may represent a quarantined family not aware that one of its members is contaminated (asymptomatic, no tests available) and therefore taking no specific actions. Case #1 is the first level of action when trying to isolate the infected people, that is, the door of his bedroom stays closed. Case #2 is one step further where the door undercut is almost completely clogged. Case #3 is similar to Case #1 but with the window partially open during the day (8:00 a.m.–6:00 p.m.) and closed at night. Finally, Case #4 is when the door undercut is 90% clogged in addition to the window opening. Simulations have been performed for two consecutive weeks in March, which was the time of the first lockup in France, with a timestep of 5 min. All ambient parameters (air temperature and relative humidity, solar radiation and wind velocity and direction) are obtained from a yearly weather file of La Rochelle (France).

Figure 2 summarizes the approach and main parameters. In addition to the containment of the infected people room, the possibility of wearing a mask by the infected and/or exposed people has also been considered. It should be noticed that the parameters regarding the source of contaminated droplets and the response of the occupants are highly variable. The set of parameters adopted represents “worst-case scenarios” as highest constant emission of contaminated droplets rate and infectivity are considered here.

### 3 RESULTS AND DISCUSSION

#### 3.1 Exposure to the contaminated droplets

Figures 3 to 7 present the 0.34 $\mu$m droplet concentration during 2 weeks in the infected people room (R3) and in the two rooms occupied by another resident, that is, room 2 (R2) during night and the living room during day (LV) for all case (#0 to #4).

In case #0 where all bedroom doors are open during day (Figure 3), the droplet concentration reaches 1000 #/m$^3$ during night and falls down to 300 #/m$^3$ when the door is open. The droplet-contaminated air leaves Room 2 to be extracted by the second floor bathroom and toilet and, for a lesser part, by the kitchen and toilet located in the first floor. As consequence, the living room always receives droplets from the stairs and its concentration is about 140 #/m$^3$. Regarding Room 2, the contaminated droplet only enters the room when its door is open. The resulting concentration is about 200 #/m$^3$. The combined effect of air renewal and droplet deposition is clearly observed in Room 2 during night.

This cycle repeats itself everyday as driven by the pressure-controlled ventilation system and the door openings. However, the effect of the wind (see Figure 8 for wind velocity and direction) is observed as the droplet concentration slightly fluctuates for most days. Note that, in the case of high wind velocities such as those occurring during Days 5 and 11–12, the fluctuations are much more noticeable.

Closing Room 3 door during day (case #1, Figure 4) induces a higher concentration in this room (1000 #/m$^3$) but limits a little the propagation to Room 2 (from 200 to 180 #/m$^3$) during day and not modifies so much the concentration in the living room. Clogging Room 3 door undercut (case #2, Figure 5) is much more effective in reducing the droplet concentration in Room 2 and in the living room. Opening Room 3 window (cases #3 and 4, Figures 6 and 7, respectively) allows the contaminated air to be partially directly extracted to outdoor without passing through the other rooms. The comparison between those two last cases shows the importance of

### Table 4 Quantum generation rate $q$ reported in literature for similar diseases and SARS-CoV-2

| Virus         | Quantum generation rate (quanta/h) | References                  |
|---------------|-----------------------------------|-----------------------------|
| Influenza     | 15–500                            | Lee et al.\textsuperscript{24}|
| MERS-CoV      | 6–140                             | WHO\textsuperscript{35}     |
| SARS-CoV-1    | 10–300                            | WHO\textsuperscript{36}     |
| SARS-CoV-2    | 14–48                             | Dai and Zhao\textsuperscript{37}|
| SARS-CoV-2    | 18.6                              | Peng et al.\textsuperscript{38}|
| SARS-CoV-2    | 32                                | Present study – resting     |
|               |                                   | source (expiration rate of   |
|               |                                   | 0.36 m$^3$/h) and $\lambda = 10$|

### Table 5 Description of each test case

| Case | Specific confinement actions |
|------|-----------------------------|
| #0   | The door of Room 3 remains open during the day (6:30 a.m.–10:00 p.m.) and closed at night |
| #1   | Room 3 door remains closed   |
| #2   | #1 + 90% of Room 3 door undercut is clogged |
| #3   | #1 + 1/3 of Room 3 window is open during the day (8:00 a.m.–6:00 p.m.) and closed at night |
| #4   | #3 + 90% of Room 3 door undercut is clogged |
clogging Room 3 door undercut to limit the droplet propagation in the whole house.

Figures 9 and 10 present the results obtained for two coarser droplets for case #0. These graphs are similar to those for smaller droplets (Figures 3 to 7). However, larger droplets are clearly confined to the second floor as deposition is much higher. Statistics for the droplet concentration in Room 3 (R3), Room 2 (R2), and in the living room (LV) are provided in Appendix as Supporting Information.
To summarize the propagation of the contaminated droplets to Room 2 and the living room, the exposures relative to Room 3 have been calculated for all droplet sizes (Figure 11). This exposure is calculated as the ratio of the 2 weeks averaged values of the droplet concentration of the selected room and Room 3. The results show that a much larger proportion of small droplets reaches the other rooms. The ratio of droplets with diameter lower than 1 µm reaching the living room is about seven times higher than the one of droplets with diameter higher than 5 µm, thanks to a smaller deposition rate for diameter around 0.5 µm. However, larger droplets carry much higher number of viruses and, even with only 1% to 10% reaching the other rooms, they remain the main propagation vector.

3.2 | Infection risk to other residents

Table 6 and Figure 12 present an estimation of the risk of illness evolution during the 14 days of recommended lockup for the
contaminated people. A simple occupancy scenario is considered to evaluate the risk of developing the disease for the other resident: The source occupant stays in Room 3 and the exposed resident is located in the living room from 6:30 a.m. to 11:30 p.m. and in the Room 2 the rest of the time. Without any action (case #0), there is a 100% risk of transmission to the other resident after only 1 week. Isolating the infected people by keeping the door closed and clogging the door undercut (case #3) induces a reduction of this risk to 61% after the same period of time. Adding to the previous case window opening during the day (case #4) reduces the risk to 39%. In this last case, the risk of illness reached 63% after 13 days, which may exceed the main contagious period, whereas it was reached in only 2 days without any action.

Another recommended action is wearing masks, for both the contaminated and the other residents. Table 7 summarizes the risk of illness with or without masks for the five studied configurations. Our results tend to show that this action cannot substitute itself to the confinement of the source occupant. Indeed, wearing masks reduces the risk of illness by 22%, whereas the strict confinement of the room 3 can reduce it by about 60%. The sum of all these precautions reduced the risk of illness at 1 week from 98% to only 17%. Figure 13 illustrates the simultaneous impact of these actions.

It is important to note that the absolute impact of the recommended actions on the risk of illness is dependent on the total dose and that this relationship is not linear. These results are, therefore, related to the test cases and the choice of parameter values, as the

**TABLE 6** Risk of illness (%) for other confined residents and for contaminated room (Room 3)

| Case | Risk of illness for other confined residents | Room 3 |
|------|--------------------------------------------|--------|
|      | Risk of illness (%) |      | Risk (%) |       |
|      | R-24h (%) | R-48h (%) | R-1w (%) | R-2w (%) | 63% Risk (h) | 63% Risk (h) |
| #0   | 44        | 67        | 98       | 100      | 43.8       | 3.2       |
| #1   | 27        | 47        | 92       | 99       | 72.5       | 3.2       |
| #2   | 12        | 21        | 61       | 84       | 179.1      | 2.9       |
| #3   | 20        | 34        | 86       | 98       | 98.6       | 3.2       |
| #4   | 5         | 10        | 39       | 65       | 318.2      | 2.9       |
infectivity. Indeed, the evolution of the risk can be considered as proportional to the dose only for very small values, and so for the very first hours of exposure. For example, if the dose is very high, the impact of the use of masks on the risk of illness can be much lower than the decrease in the virus intake. In order to illustrate this observation, Figure 14 summarizes the impact of the recommendations on the dose. For instance, in the case #4, if both the source occupant and the other residents are masked, these results show that 95% of the exposition can be avoided. In comparison, the risk of illness is reduced by 83% at 1 week and 67% at 2 weeks in this configuration.

3.3 | Variants, vaccination, and sensitivity of this model

This approach documented the relative impacts of classic recommendations to limit the contagion of occupants. As previously noted, our model is dependent on the ratio of two main parameters: the viral load in the emitted droplets and the infectivity ($\alpha / \lambda$) (Equations 1 and 2). These parameters were particularly challenging to estimate and have a direct impact on the calculation of the risk of illness from a given dose.

Moreover, the infectivity is directly influenced by the capacity of the virus to survive and enter human cells. Both the mutations of variants and the efficiency of the immune system, impacted by the vaccination, can therefore affect our results.

The current data on pandemic of SARS-CoV-2 show the emergence of more contagious coronavirus is a reality, such as the recent Delta variant. Teyssou et al.\textsuperscript{33} showed a median viral load at least ten times higher than Beta and historical SARS-CoV-2 variants. On the contrary, vaccination should reduce both the viral load and the infectivity.

In order to illustrate the complexity induced by the lack of data on the estimation of the ratio ($\alpha / \lambda$), the mutations of the virus and

![Figure 12](image1.png)

**FIGURE 12** Risk of illness for the confinement actions for a 14 days period

![Figure 13](image2.png)

**FIGURE 13** Impact of the use of masks on the risk of illness for two configurations: Case #0 and Case #4

| Case | Risk of illness at 1 week (%) | Risk of illness at 2 weeks (%) |
|------|-----------------------------|-----------------------------|
| #0   | 98                          | 78                          |
| #1   | 92                          | 72                          |
| #2   | 61                          | 37                          |
| #3   | 85                          | 62                          |
| #4   | 38                          | 22                          |

(1 - $M_s$) represents the efficiency of the mask for the source occupant and (1 - $M_e$) the efficiency for the exposed resident.

![Table 7](image3.png)

**TABLE 7** Impact of masks worn by the source occupant or the other residents on the risk of illness (%)
the growth of vaccination, a sensitivity analysis of this model has been carried on. It should enlighten the absolute results previously discussed. It has been chosen to explore two orders of value only, from $\alpha_{cv}/\lambda = 10^7$ mL$^{-1}$ to $\alpha_{cv}/\lambda = 10^9$ mL$^{-1}$, as $\alpha_{cv}/\lambda = 10^8$ mL$^{-1}$ is the value previously adopted. The purpose is illustrative as the true uncertainties are much higher.

Table 8 summarizes the sensitivity of the model on the ratio $\alpha_{cv}/\lambda$ for case #0. Figures 15 and 16 illustrate the impact of the uncertainties on the ratio $\alpha_{cv}/\lambda$ on the evolution of the risk of illness in cases #0 and #4.

The impact of a new variant or the vaccination of an occupant can therefore be predominant. It is nevertheless, in this work, impossible to estimate it precisely. For example, our results show a very large scale of risk, from 5% to 99%, in case 4 at 1 week. These results illustrate the need of much more precise inputs for the use of a dose-response model. More residential epidemiological studies and laboratory data should be valuable to improve the quality of the results calculated with this model in reducing uncertainties on its parameters. Furthermore, the results are limited to the specific building and scenarios adopted in the present study and thus are related to only...
one building geometry, one ventilation system, one climate, and period of time. Still, the insights on the relative impacts of confinement actions and on the wearing of masks remain likely consistent.

4 | CONCLUSIONS

The results of the five worst-case scenarios modeled in the present study show that there is a high risk of virus transmission via air transport to other residents of the dwelling if there is already an infected person inside. Yet, this study proves most recommended actions have a positive impact to preserve the health of the exposed confined occupants.

A multizone approach with a distinction of several size bins for contaminated droplets has shown several advantages and was favored using a dose–response model. However, the risk of illness calculation relies on a pool of coefficients that are difficult to estimate. The infectivity was particularly challenging, and we chose to present the results obtained for its highest estimation. Epidemiological data argue for this choice; nevertheless, this parameter has a direct impact on the exposure; and therefore, we should remain prudent on the absolute results of the risk of illness obtained here.

The insights on the relative impacts of confinement actions and on wearing of masks remain likely consistent. Residential epidemiological studies could be valuable to improve the quality of this model in reducing uncertainties on its parameters.

NOMENCLATURE

| Symbol | Parameter (Unit) |
|--------|-----------------|
| $C_{\text{copies}}$ | Concentration of virus copies for a size bin $i$ (copies/m$^3$) |
| $C_v$ | Viral load in sputum (copies/ml) |
| $d$ | Dose, quantity of copies inhaled by an occupant (copies) |
| $IR$ | Inhalation rate (m$^3$/h) |
| $M_E$ | Penetration factor of the mask worn by the exposed occupant. $(1 - M_E)$ is the efficiency of the mask worn by the exposed occupant (-) |
| $M_S$ | Penetration factor of the mask worn by the contaminated occupant. $(1 - M_S)$ is the efficiency of the mask in reducing the virus emission from the contaminated occupant (-) |
| $N_i$ | Concentration of aerosol droplets of the size bin $i$ (particles/m$^3$) |
| $q$ | Quantum generation rate of the source (copies/h) |
| $Q$ | Volumetric airflow rate (m$^3$/h) |
| $R_d$ | Risk of illness at a dose $d$ (probability between 0 and 1) (-) |
| $RDF_i$ | Respiratory deposition fraction for the size bin $i$ (-) |
| $S_{\text{copies}}$ | Source of virus copies for a size bin $i$ (copies/h) |
| $S_{\text{part}}$ | Estimated source of particles for a size bin $i$ (particles/h) |
| $V_i$ | Volume of one aerosol droplet of the size bin $i$ (m$^3$) |
| $\alpha$ | Concentration factor of aerosol droplets (-) |
| $\lambda$ | Infectivity (-) |

PEER REVIEW

The peer review history for this article is available at https://pubons.com/publon/10.1111/ina.13035.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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SUPPORTING INFORMATION
Additional supporting information may be found in the online version of the article at the publisher’s website.

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