Indoor transmission of airborne viral aerosol with a simplistic reaction-diffusion model

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Abstract A simplistic reaction-diffusion model is undertaken in the present work to mathematically explore the spatio-temporal development of concentration of indoor aerosols containing infectious COVID-19 respiratory virus nuclei. Extracting exact solutions of concentration field under the influence of several physical parameters is preferred rather than adopting a more realistic complex model requiring time-consuming numerical simulations. Even though the proposed model is not sophisticated, the analytical solutions can provide quick prediction of the probability of contracting the virus in a ventilated closed room. Moreover, from the obtained elementary solutions of the viral concentration field, it is easy to analyze its spatio-temporal evolution and final equilibrium state. Formulae enable us to estimate the time to get infected and the risk of getting infected within an elapsed time under various physical operative situations involving a uniform infectious particle mixture ejection into the medium, wearing a face mask with a well-defined efficiency parameter and taking into account a localized source of infection. One of the essential conclusion from the current research is that less aerosols carrying COVID-19 particles are as a result of good indoor ventilation conditions, of removing the medium air through windows (or other exits) and of wearing masks of high efficiency. Moreover, the risk and probability of being caught by the indoor COVID-19 disease increases in time, particularly in the downstream of a localized infectious person. The results can be beneficial to understand and take necessary safety considerations against the infection risk in closed public or governmental environments.

1 Introduction

Since the outbreak of COVID-19, the pandemic conditions worldwide are getting worse and the indoor activities turn out to be more risky due to the airborne transmission of virus particles (aerosol) emitted by infectious people in room [1,2]. Clear evidences of virus airborne via respiratory droplets and viable survival in air can be referred to [3–5]. Ventilation [6,7] by air-conditioners or by releasing out the polluted air through windows in closed rooms is thus life saving, among other precautions [8]. The present effort is to formulate such physical effects into a simple reaction-diffusion model to analytically elaborate the spatial and temporal spread of indoor concentration air-suspended viruses.

Aerosols due to COVID-19 respiratory particles and their transport through air in closed spaces have been investigated recently in view of experimental and computational aspects [9]. A case study was reported regarding the air in hospital room with COVID-19 patients in [10]. Superspreader events of COVID-19 taking place indoors were warned the world in the analysis in [11]. It was demonstrated in [12] that infectivity pathogens can be transported over extended distances with airflows in indoor. Interactions of coughing and sneezing droplets of coronavirus disease with indoor air in an African hospital ward were measured in [13]. The route to coronavirus disease as a consequence of the outdoor airborne transmission between neighboring apartments in high density cities was examined in [14]. The good/poor ventilation and particle emission rates were computationally simulated in [6,7] to predict the amount of adequate indoor concentration for someone to get infected or else to avoid from the risky amount. An overview of ventilation influences on the indoor spread of COVID-19 was given in [15]. It was proven in [16] that the rate of inhaled/exhaled virus transmission can be reduced by continuous usage of appropriate face masks. Experimental conducts in [8] exhibited that in a relatively large indoor environment the common face masks provide effective protection under certain circumstances, despite the fact that the exhalation filtration efficiency is much lower than the ideal filtration efficiency of the mask material. In a poorly ventilated restaurant, the airborne transmission of SARS-CoV-2 droplets was simulated in [17] with some suggestions. Airborne pathogen transport and potential risks were...
analyzed in the numerical modelling in [18]. The prevention of COVID-19 transmission was advised from the numerical simulation in [19] incorporating social distance and ventilation effects.

The aforementioned experimental and numerical simulations to search for the mechanisms of airborne COVID-19 are absolutely valuable, though they require robust and modern infrastructures owing to the model complexity and expensive to implement. The motivation in the present endeavour is to present a simple reaction-diffusion model from which notably quick analysis can be accessed. For this purpose, a simplistic reaction-diffusion model is developed from which the airborne transmission of air-suspended viral particles can be exactly formulated through a concentration function. Hence, the effects of air-conditioning, Peclet number, wearing different masks, isolated or uniform source and air freshening by windows in indoor activities can be studied on the spatio-temporal distribution of concentration aerosols. Thanks to such formulae, the infection risk and the time to get contagious disease can be estimated, which are deadly important in controlling the spread of COVID-19 in closed public or governmental environments. The current conclusions bring the useful information justifying the earlier computations in the literature that higher ventilation reduces the concentration of virus droplets and people who are exposed to higher concentrations of virus or for longer periods of time are more likely to be infected.

2 Reaction-diffusion model with advection

We consider the time evolution and spatial distribution of concentration of airborne infectious droplets (aerosol) generated by expiratory activity diseases within a closed indoor as sketched in Fig. 1. A few assumptions are made to constitute the mathematical model. For instance, the concentration distribution of aerosol in the room (such as classroom), for instance due to COVID-19, is advected along \( X^- \) axis only assuming a longer horizontal \( y^- \) axis perpendicular to \( X^- \) axis. The virus particles are presumed to propagate with a constant airflow velocity \( U \) owing to the ventilation by air-conditioning. The attention should be drawn that, the horizontal uniform flow assumption is only applicable to a very special case of ventilation since buoyancy driven upward flow is predominant in more realistic cases. Letting also \( t \) be the time, \( C \) be the concentration of viral aerosol, \( k \) be the diffusion coefficient, \( R \) be the uniform infectious particle injection rate, \( \lambda \) be the particle decay rate/air exchange rate, the advection-diffusion model governing the COVID-19 virus concentration can be formulated through the system [15,20]

\[
C_t + UC_x = kC_{xx} - \lambda C + R, \\
C(X, 0) = 0, \\
kC(0, t) = 0, \quad kC(L, t) = -a. \tag{1}
\]

No initial infectious aerosol concentration is implied by (1). Moreover, no particles can escape through the wall at \( x = 0 \), but, only there is a leakage of the rate \( a \) through the windowed wall at \( x = L \) [21]. Another choice would be to apply appropriate boundary conditions in a manner that all air moves out with velocity \( U \) on account of mass conservation. In model (1), the ventilation impact is simply represented by the sink term \(-\lambda C\). The source of viral particles is due to an emitted uniform and continuous injection rate \( R \) [22]. It is further remarked that more mathematical complexity can be easily brought into the model in (1), which is avoided here for the sake of obtaining exact concentration solutions from which qualitative particle concentration, and its spatial and temporal development can be conceived. This is the preferred ultimate choice here although the author is aware of the fact that the ability to obtain exact solutions can not always be the deciding factor in determining the model of choice when describing a physical problem.

With the assist of the subsequent transformations

\[
\Lambda = \frac{\lambda L^2}{k}, \quad \text{Pe} = \frac{UL}{k}, \\
X = Lx, \quad t = \frac{L^2}{k}\tau, \\
C = \frac{RL^2}{k}c, \quad \alpha = \frac{a}{RL}. \tag{2}
\]

one can recover the dimensionless advection-diffusion system from (1)

\[
c_\tau + \text{Pe}c_x = c_{xx} - \Lambda c + 1, \\
c(x, 0) = 0, \\
c_x(0, \tau) = 0, \quad c_x(1, \tau) = -\alpha, \tag{3}
\]

where the model is seen to be governed by three physical parameters; namely, the Peclet number \( \text{Pe} \), the
exchange rate of room air $\Lambda$ and the flux of leakage $\alpha$ by means of windows.

The total amount of inhaled air cloud containing the infectious viral particles by someone within the time period $[0, t]$ can be computed from [23]

$$d(x, t) = \int_0^t \rho C dt,$$  (4)

where $\rho$ is the rate of breathing. If $P$ shows the total inhaled airborne virus particle dose which is critical to become infected at the time $t_i$, the dimensionless time $\tau$ to be infected by the aerosol cloud can be estimated from [24]

$$P = p_0 \int_0^\tau \rho C d\tau,$$  (5)

with $p_0$ being the dimensionless breathing factor as a result of transformations (2). By the help of (4), the probability of being infectious can also be defined by means of an exponential probability density function [25] in the following manner

$$p(x, t) = 1 - e^{-p_1 d(x, t)},$$  (6)

where $p_1$ is a rate factor of infectious disease under consideration.

### 3 Analytical analysis

The airborne transfer of infectious particles takes place under steady-state equilibrium conditions in the long run by solving the time-independent system

$\text{Pec}' = e'' - \alpha c + 1, \quad c'(x = 0) = 0, \quad c'(x = 1) = -\alpha.$  (7)

The concentration solution of particles from (7) is found to be

$$c(x) = \frac{-2 + 2Pe_1 + e^{\frac{1}{2}(Pe_1)(1-x)}\alpha (Pe - Pe_1) - e^{\frac{1}{2}(x-1)(Pe_1)}\alpha (Pe + Pe_1)}{2(-1 + e^{Pe_1})\Lambda},$$  (8)

where $Pe_1 = \sqrt{Pe^2 + 4\Lambda}$. It is noted that in the limit of $\alpha \to 0$, that is when no airborne particles are allowed through windows, Eq. (8) yields solely the uniform equilibrium

$$c(x) = \frac{1}{\Lambda},$$  (9)

which implies physically that the concentration of aerosol and the reduction rate is inversely proportional, as expected. Intriguingly, the same limit in (9) is attained in the limit of large Peclet number, too. Otherwise, the accumulated COVID-19 aerosol at the walls are determined from (8)

$$c(0) = \frac{1 - e^{\sqrt{Pe^2 + 4\Lambda}} + e^{\frac{1}{2}(-Pe + \sqrt{Pe^2 + 4\Lambda})\alpha \sqrt{Pe^2 + 4\Lambda}}}{\Lambda - e^{\sqrt{Pe^2 + 4\Lambda}}},$$

$$c(1) = \frac{2 + Pe\alpha - \sqrt{Pe^2 + 4\Lambda} \coth \left(\frac{1}{2}\sqrt{Pe^2 + 4\Lambda}\right)}{2\Lambda}.$$  (10)

If the instantaneous distribution of exhaled aerosols in the room air negligibly varies along spatial positions (as a first approximation), as can be inferred from the experimental study of [8], which is particularly true when $\alpha$ is small in system (3), the temporal evolution of $c$ can be approximated from the simplified linear ordinary initial value problem

$$c(\tau) = 1 - \Lambda c, \quad c(\tau = 0) = 0,$$  (11)

whose solution can be expressed as

$$c(\tau) = \frac{1 - e^{-\Lambda\tau}}{\Lambda}.$$  (12)

Unlike the above, the general time-dependent concentration should be sought upon solving the full time-dependent spatial system (3). Separation of variables or Laplace transform seems to be ready useful tools to access the analytical solution; here the Laplace transform is preferred. Hence, applying the Laplace transform to (3) with the Laplace variable $s$ by virtue of

$$L(c(\tau, x)) = F(x, s),$$

we obtain the Laplace transformed solution of concentration

$$F(x, s) = \frac{2 - e^{-\frac{1}{2}(p-\sqrt{Pe})(1-x)}\alpha (p + pex (p-Pe)+Pe)}{2s(s + \Lambda)},$$  (13)

where $p = \sqrt{Pe^2 + 4(s + \Lambda)}$. To invert (13) and get the physical solution $c$, we employ the inverse Laplace transform and ultimately make use of the residue theorem.
$$c(\tau, x) = \text{Res}_{s = 0} + \text{Res}_{s = -\Lambda} + \sum_{k=0}^{\infty} \text{Res}_{s = s_k},$$  

(14)

where $s_k = \frac{1}{2} (-\text{Pe}^2 - 4\Lambda - 4\pi^2 k^2)$ are the simple poles from the solution of the denominator in (13)

$$-1 + e^{\sqrt{\text{Pe}^2 + 4(s + \Lambda)}} = 0.$$  

(15)

Upon carrying out the calculations, the first part of the residue solution in (14) at $s = 0$ gives literally rise to the steady state solution in (8). Following this, the residue calculation at $s = -\Lambda$ yields,

$$\text{Res}_{s = -\Lambda} = \frac{e^{-\Lambda \tau} (1 - e^{\text{Pe} + \text{Pe} \alpha})}{(-1 + e^{\text{Pe}) \Lambda}},$$

(16)

and eventually, the residues at the zeros of equation (15) result in the solution

$$\sum_{k=0}^{\infty} \text{Res}_{s = s_k} = \sum_{k=0}^{\infty} 16(-1)^k e^{\frac{1}{2}\text{Pe}(-1 + x)} e^{-\frac{1}{2}(\text{Pe}^2 + 4(k^2 \pi^2 + \Lambda))} k\pi\alpha(2k\pi\cos(k\pi x) - \text{Pe}\sin(k\pi x)) / (\text{Pe}^2 + 4k^2\pi^2) (\text{Pe}^2 + 4(k^2\pi^2 + \Lambda)).$$

(17)

Consequently, thanks to the full solution in (14) with consideration of (8), (16) and (17), it is possible to achieve the exact representations for the time/probability to get infection making use of the relations in (4–6).

### 4 Results and discussions

According to the data available in the open literature, very poor, poor, pre-pandemic recommended and pandemic-updated recommended ventilation scenarios can be represented by the typical parameter values as listed in Table 1, refer to [26–28] for such ventilation settings and appropriate eddy diffusion coefficients. Therefore, the physical parameter range of $\Lambda$ is taken as $[2, 3]$, whereas those of $\text{Pe}$ and $\alpha$ as $[0, \infty)$ in the present conduction.

Initially, the impacts of physical parameters $\Lambda$, $\text{Pe}$ and $\alpha$ on the time-independent spatially distributed concentration of virus accumulation are demonstrated in Fig. 2a–c, as computed from (8). It is clear from Fig. 2a that poor ventilation is the main source of high risk of inhaling more viral concentration. The accumulated virus particle cloud is reduced considerably by improving air exchange rates. The concentration of virus particles is also lowered with small Peclet numbers as compared to the higher concentrations corresponding to the higher Peclet numbers. Thus, it is deduced that not speedy recirculation or higher eddy diffusion leading to small Peclet numbers is favourable, owing to the fact that a rapid diffusion of viral particles in the air takes place. This is also relevant to the good ventilation cases as realized from Table 1. In parallel to the physical intuition, the presence of outward flux of diseased droplets adjacent the windows at $x = 1$ is a beneficial route to reduce the concentration of infectious aerosol, as visualized in Fig. 2c. As a result, it is observed from Fig. 2a–c that concentration is less adjacent open windows at $x = 1$ (with outward flux of unity) rather than the far wall at $x = 0$ where the air flows through vents.

This result is compatible with the data of hospitals in Wuhan [29]. Notice that the large/small limiting behavior of $\text{Pe}$, $\alpha$ given in formula (9) is captured well in Fig. 2a–c.

The accumulated aerosol concentrations on the walls are revealed next in Fig. 3a, b with varying $\alpha$ and $\Lambda$. An effective way of removing the viral concentration at the far wall (and in spatial direction naturally) is shown to be with higher air exchange rates only, not affected by the flux parameter, see Fig. 3a. On the other hand, both factors of $\alpha$ and $\Lambda$ are effective in decreasing the amount of concentration adjacent the windowed wall, see Fig. 3b. Actually, without outward flux with $\alpha = 0$, the jet of airflow by air conditioner carries the aerosol from one wall to the other in a recirculating loop in a closed room.

Figure 4 depicts the time evolution of virus particle concentration from the predictive non-spatially varying formula (12). Again in line with the expectation, the concentration develops from the initial startup and quickly reaches its steady-state with shorter time and

| Condition             | $\Lambda$     | $L$ | $k$        | $U$   | $\Lambda$ | $\text{Pe}$ |
|-----------------------|---------------|-----|------------|-------|------------|-------------|
| Very poor             | $3.3 \times 10^{-5}$ | 8   | $8.8 \times 10^{-4}$ | 0.15  | 2.40       | 1363.64     |
| Poor                  | $2.0 \times 10^{-4}$ | 8   | $5.3 \times 10^{-3}$ | 0.15  | 2.415      | 226.415     |
| Pre-pandemic          | $8.3 \times 10^{-4}$ | 8   | $2.2 \times 10^{-2}$ | 0.15  | 2.414      | 54.5455     |
| Pandemic-updated      | $1.7 \times 10^{-3}$ | 8   | $4.4 \times 10^{-2}$ | 0.15  | 2.473      | 27.2727     |
Fig. 2  Steady spatially distributed concentration of aerosol in indoor. Impacts of (a) $\Lambda$ at $\text{Pe} = 10$ and $\alpha = 1$, (b) $\text{Pe}$ at $\Lambda = 2.4$ and $\alpha = 1$, and (c) $\alpha$ at $\Lambda = 2.4$ and $\text{Pe} = 15$.

Fig. 3  Steady distributed concentration of aerosol in indoor at the walls. (a) $c(0)$ and (b) $c(1)$. 
Unsteady concentration of aerosol in indoor from the linear equation in (12) with various $\Lambda$.

Final smallest concentration size in the good ventilation conditions. Unlike to this, the settlement to equilibrium is considerably delayed with final higher concentrations in the absence of a good ventilation. Such behaviors with or without ventilation in a COVID-19 infectious room were observed in the experimental performance in [8] (see figures 6 through 9 therein) as well as in numerical simulations of [6] (see figure 3 therein).

On account of the exact concentration solution in (12), the time to get infected for an indoor person can be calculated from (5) as

$$\tau_i = \frac{1 + P/p_0 \Lambda^2 + W\left(-e^{-1-P/p_0 \Lambda^2}\right)}{\Lambda},$$

(18)

where $W(x)$ is the principal branch of the Lambert function. Figure (5) displays the critical dimensionless time against $\Lambda$ for a particular value of $P/p_0 = 10$. Changing this value has not altered the trend of figure, except the increment in the critical time. It is unsurprisingly concluded that the less the air exchange rate with poor ventilation in the room is, the shorter to become infected is. A linear relationship occurs between the infection time $\tau_i$ and air exchange rate $\Lambda$.

Again with the help of the solution in (12), the probability of getting infected in time can be computed from (6) in the form

$$p(\tau) = 1 - e^{-\frac{p_1(-1+e^{-\tau \Lambda+\tau \Lambda_1})}{\Lambda^2}}.$$  

(19)

In Fig. 6, the probability of becoming infected is visualized against $\Lambda$ at $p_1 = 10^{-2}$ evaluated from (19). Expectedly, the more someone to get exposure to the polluted air concentration, the risk is greatly increasing with enhanced probability, whereas this is shifted down in the case of good ventilated rooms. If the probability is required to be less than 50%, the reasonable duration of gathering in the room should not exceed roughly $150 < \tau < 230$, strictly depending on the values of $\Lambda$. Hence, Fig. 6 is quite useful to determine the evacuation time of the room so that a safe reset of the viral concentration can be fulfilled.

Spatio-temporal development of airborne COVID-19 particle concentration are exhibited in Fig. 7a, b for $\Lambda = 2.4, \alpha = 1$ and $Pe = 3.5$. Before settling down to the final steady-state solution given by (8), an abrupt change is anticipated close to the window zone where concentration flux is permitted to flow outwards. It is worthy to mention that the series in (17) is convergent for adequately small number of terms, such as $M = 5$ assigned to produce Fig. 7a, b (and for the rest). In order to justify this assertion, Fig. 8a, b are drawn with $M = 0$ and $M = 1$ for $\Lambda = 2.4$, $Pe = 10$ and $\alpha = 1$, which shows an indistinguishable resemblance. As a result, even retaining one or two terms in series solution (17) are adequate to satisfactorily capture the concentration evolution, that is a uniformly true assessment. Hence, an accurate representation can be formulated with truncation $M = 1$.
The final figures in Fig. 9a–c are to visualize the probability of getting infected in 3D-perspective for $\Lambda = 2.4$, $Pe = 3$, $\alpha = 1$, and $p_1 = 10^{-2}$ and $\Lambda = 2.4, 2.6, 2.8$ computed from (6). In line with the previous findings as seen from Fig. 6, the probability and hence the risk is increasing in time. Moreover, the people sitting away from the windows are prone to the higher probability exposed to more pollutant air in the case of poor ventilation. Naturally, the contagion risk is increasing in time in the closed spaces.

Eventually, adapted from the idea in [7], the emission rate $R$ of airborne particles in system (1) from an infected mankind can be regularized by wearing a mask with efficiency $\eta$, such that now, $R$ is given by

$$ R(1 - \eta). $$

In view of (21), no mask corresponds to $\eta = 0$ as studied so far within the present research, but increasing $\eta$ ($< 1$) means wearing an effective mask emitting less.
viral particles. In this case, all the formulae derived so far will involve \( \eta \) and hence undoubtedly the particle concentration and airborne disease transmission are effectively reduced. Indeed, under (21), model and formula in (11–12) turn into

\[
c_{\tau} = 1 - \eta - \Lambda c, \quad c(\tau = 0) = 0,
\]

whose solution is now given by

\[
c(\tau) = (1 - \eta) \frac{1 - e^{-\Lambda \tau}}{\Lambda}.
\]

Fig. 9  Probability to get infected for \( \text{Pe} = 3, \alpha = 1 \) and \( p_1 = 10^{-2} \). a \( \Lambda = 2.4 \), b \( \Lambda = 2.6 \) and c \( \Lambda = 2.8 \)

Likewise, the uniform emission/spread rate of infection throughout the spatial domain \( x \in [0, 1] \) can be replaced by a localized infectious source at the mid-point \( x = 1/2 \) (though not obliged), which modifies the reaction-diffusion model (3) as

\[
c_{\tau} + \text{Pe} c_{x} = c_{xx} - \Lambda c + \delta(x - 1/2),
\]

\[
c(x, 0) = 0, \quad c_{x}(0, \tau) = 0, \quad c_{x}(1, \tau) = -\alpha,
\]

where localized infectious source is represented by the distribution function \( \delta(x - 1/2) \). The steady-state solution corresponding to system (24) is deviated from (8) and given now in the form

\[
c(x) = \frac{1}{2(-1 + e^{\text{Pe}_1})} \frac{e^{-\frac{1}{2}(-2\text{Pe} - \text{Pe}_1)}}{\Lambda \text{Pe}_1} e^{\frac{1}{2}(-2\text{Pe} - \text{Pe}_1)} \left( -2e^{\frac{1}{2}(\text{Pe} + 2\text{Pe}_1 - 2x\text{Pe}_1)} (-1 + e^{\text{Pe}_1}) \right)
\]
which takes a non-uniform shape dissimilar to (9) when \( \alpha \) is set to zero

\[
\begin{align*}
  c(x) &= \frac{1}{2 (1 + e^{Pe_1}) A Pe_1} \\
  &\quad \times \left( 2 e^{Pe_1} \Lambda + 2 e^{(1+x) Pe_1} \Lambda + e^{Pe_1} (Pe^2 + 2\Lambda) \right) \\
  &\quad + e^{\frac{1}{2} (1+2x) Pe_1} (Pe^2 + 2\Lambda) + \left( e^{Pe_1} - e^{x Pe_1} \right) \\
  &\quad \times \left( 2 (-1 + e^{Pe_1}) \Lambda H (-1 + 2x) + e^{x Pe_1} Pe_1 \right) \right)
\end{align*}
\]

(25)

In (25–26) \( H \) represents the Heaviside function. The steady concentration at the walls are now given by

\[
\begin{align*}
  c(0) &= \frac{e^{\frac{1}{2} (-2 Pe + Pe_1)} \left( \left( e^{Pe/4} + e^{\frac{1}{2} (Pe + 2 Pe_1)} - 2 e^{Pe_1} \alpha \right) Pe_1 + e^{Pe/4} \left( -1 + e^{Pe_1} \right) Pe \right)}{2 (-1 + e^{Pe_1}) \Lambda} \\
  c(1) &= \frac{e^{\frac{1}{2} (Pe + 3 Pe_1)} \left( Pe_1 - Pe \right) + e^{Pe_1} \alpha \left( -Pe_1 + Pe \right) + e^{\frac{1}{2} (Pe + Pe_1)} \left( Pe_1 + Pe \right) - \alpha (Pe_1 + Pe)}{2 (-1 + e^{Pe_1}) \Lambda}.
\end{align*}
\]

(27)

The impacts of localized infectious source on the steady-state concentration distribution are summarized in Fig. 10a–d. The build-up of concentration field from the location of infected person is clearly visualized from these figures relying upon the ventilation, Peclet number and strength of outward flux through window. It is remarked that the progress of concentration field is very much different from those figures in Figs. 2, 3 and 4. Figure 10a–d reveal that the concentration which is the highest at the position of infectious person spreads downwards and upwards from the mankind, remaining bigger at the downwind directions due to the drifting effect by airflow of vents, which is ultimately lowered by the action of outward air flux through the windowed walls. It is easy to deduce that the risk of getting infected by the people sitting downward locations from the source will be naturally higher than the other locations. Such observations were also made in the 2D numerical simulations in [6, 7] as well as in hospital wards in Wuhan [29]. So, the public indoor environments like restaurants, offices, hospitals and classrooms should seriously take into account this physical evidence.

5 Conclusions

The assessment of concentration distribution of viral particles from aerosols due to airborne COVID-19 viruses within closed spaces through a reaction-diffusion mathematical model is the main motivation of the current paper. Not to complicate the formulation and for the sake of obtaining the analytical solutions in elementary form, a simplified model is adopted, in which the spread of Covid-19 in a room with ventilation on one side and an open window on the other is modelled using a one-dimensional advection-reaction-diffusion equation.

Having solved the constructed diffusion model, concentration solutions are initially derived. Hence, spatiotemporal evolution of concentration and final steady-state development are clarified. An in-depth analysis is then implemented regarding the physical situations, including the uniform infectious particle mixture ejection into the medium, the wearing a face mask with a well-defined efficiency parameter and the introducing a localized source of infection. By the help of the presented formulae, the time to get infected and the infection probability can be easily estimated and the exposure to airborne transmission can be overwhelmed.

The fundamental outcomes from the current mathematical effort are that indoors with good ventilation conditions expectedly accumulate less viral concentration, which can be further reduced by exiting the medium air through windows (or windowed walls) as well as by wearing masks of high efficiency. Additionally, the risk and probability of becoming infected indoor rise as time elapses and the downstream of a localized infectious person is more risky than the other spatial locations, unless sufficient outward flux of polluted air through windows is present within the room.

It is believed that the simplistic reaction-diffusion model of airborne infectious virus concentration whose results are presented here can be updated with more physical parameters of a special disease. Although the handled model is simple, it will be at the disposal of more realistic and complex models recently being developed, accounting for the higher dimensions, diff-

\[
\begin{align*}
  c(x) &= \frac{1}{2 (1 + e^{Pe_1}) A Pe_1} \\
  &\quad \times \left( 2 e^{Pe_1} \Lambda + 2 e^{(1+x) Pe_1} \Lambda + e^{Pe_1} (Pe^2 + 2\Lambda) \right) \\
  &\quad + e^{\frac{1}{2} (1+2x) Pe_1} (Pe^2 + 2\Lambda) + \left( e^{Pe_1} - e^{x Pe_1} \right) \\
  &\quad \times \left( 2 (-1 + e^{Pe_1}) \Lambda H (-1 + 2x) + e^{x Pe_1} Pe_1 \right) \right)
\end{align*}
\]

(25)
ferent material face mask wearing, filtration efficiency, social distancing, number of localized infected people in the enclosed, more complex indoor structures, inhaling/exhaling rates and ventilation conditions, gravitational settling of particles in time, virus inactivation, some of which can be inferred from the recent researches in [6–8,30–32].

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