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Visualization of the infection risk assessment of SARS-CoV-2 through aerosol and surface transmission in a negative-pressure ward

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1. Introduction

Since December 2019, coronavirus disease (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has become a great challenge to the world’s public health system. Nosocomial infections have occurred frequently in medical institutions worldwide during this pandemic. Thus, there is an urgent need to construct an effective surveillance and early warning system for pathogen exposure and infection to prevent nosocomial infections in negative-pressure wards. In this study, visualization and construction of an infection risk assessment of SARS-CoV-2 through aerosol and surface transmission in a negative-pressure ward were performed to describe the distribution regularity and infection risk of SARS-CoV-2, the critical factors of infection, the air changes per hour (ACHs) and the viral variation that affect infection risk. The SARS-CoV-2 distribution data from this model were verified by field test data from the Wuhan Huoshenshan Hospital ICU ward. ACHs have a great impact on the infection risk from airborne exposure, while they have little effect on the infection risk from surface exposure. The variant strains demonstrated significantly increased viral loads and risks of infection. The level of protection for nurses and surgeons should be increased when treating patients infected with variant strains, and new disinfection methods, electrostatic adsorption and other air purification methods should be used in all human environments. The results of this study may provide a theoretical reference and technical support for reducing the occurrence of nosocomial infections.

Keywords:
- Aerosol and surface transmission
- Visualization of infection risk assessment
- Negative-pressure ward
- Air changes per hour
- Variant of concern

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ABSTRACT

Since December 2019, coronavirus disease (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has become a great challenge to the world’s public health system. Nosocomial infections have occurred frequently in medical institutions worldwide during this pandemic. Thus, there is an urgent need to construct an effective surveillance and early warning system for pathogen exposure and infection to prevent nosocomial infections in negative-pressure wards. In this study, visualization and construction of an infection risk assessment of SARS-CoV-2 through aerosol and surface transmission in a negative-pressure ward were performed to describe the distribution regularity and infection risk of SARS-CoV-2, the critical factors of infection, the air changes per hour (ACHs) and the viral variation that affect infection risk. The SARS-CoV-2 distribution data from this model were verified by field test data from the Wuhan Huoshenshan Hospital ICU ward. ACHs have a great impact on the infection risk from airborne exposure, while they have little effect on the infection risk from surface exposure. The variant strains demonstrated significantly increased viral loads and risks of infection. The level of protection for nurses and surgeons should be increased when treating patients infected with variant strains, and new disinfection methods, electrostatic adsorption and other air purification methods should be used in all human environments. The results of this study may provide a theoretical reference and technical support for reducing the occurrence of nosocomial infections.

1. Introduction

Since December 2019, coronavirus disease (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has become a major global public health issue that has had a profound impact on social development worldwide and continues to pose a great challenge to the world’s public health systems. To prevent the spread of COVID-19, governments around the world have taken a series of measures, including monitoring, diagnosis, treatment, and emergency prevention and control in public areas (Li et al. 2020). Negative-pressure wards have become the main battlefield in the fight against COVID-19. As the global pandemic continues, the prevention and control of infection in medical institutions has become extremely important.

Nosocomial infections have occurred frequently worldwide during this pandemic. As of February 24, 2020, 3387 (4.4%) of the 77,262 COVID-19 cases in China were medical workers in healthcare facilities (Zhan et al. 2020). On February 7, 2020, Zhongnan Hospital of Wuhan University reported that 28 confirmed cases had occurred in the hospital itself since January 1 (Wang et al. 2020). The proportion of nosocomial infections in the hospital was up to 41.3%, and nearly 29% of the cases were among medical staff (Wang et al. 2020). As of March 22, 2020, 4826 medical workers in Italy had been infected, representing 9% of the total number of confirmed COVID-19 cases in this country. Additionally, the Ministry of Health in Spain counted 42,111 cases of COVID-19 among medical providers since May 3, 2020, representing 19% of the country’s total (Xiang et al. 2020). It is imperative to establish a monitoring and early warning system for negative-pressure wards that not only protects medical staff from pathogen infection but also ensures the...
continuity of medical services and provides strong support for the prevention and control of COVID-19.

Respiratory infections can spread through multiple routes, making infection control in negative-pressure wards challenging. Aerosols, droplets and contact have been identified as the main transmission routes for SARS-CoV-2 (Arons et al. 2020; Canova et al. 2020; Zhou et al. 2020). In addition, a large number of nosocomial infections have developed through noncontact routes during the COVID-19 pandemic, suggesting that aerosols are likely to be a crucial transmission route for SARS-CoV-2. Aerodynamic studies of SARS-CoV-2 have suggested that the exposure risk for COVID-19 needs to be further studied. During the prevention and control of COVID-19, medical staff are often exposed to large amounts of aerosols from patients while administering treatment options such as sputum aspiration and intubation. Our previous study at Wuhan Huoshenshan Hospital from February to March 2020 found that SARS-CoV-2 was widely distributed in the air and on object surfaces in the intensive care unit (ICU) and general COVID-19 ward (GW). Even in an ICU with a functioning ventilation system, viral aerosols can be detected up to a maximum transmission distance of 4 m (Guo et al. 2020). Lan et al. conducted an aerodynamic analysis of SARS-CoV-2 in two hospitals in Wuhan and found a high concentration of SARS-CoV-2 nucleic acid in the toilets used by patients and a high concentration of viral RNA in the medical staff area (Li et al. 2020). Ge et al. assessed the exposure risk of SARS-CoV-2 in hospital settings and found that SARS-CoV-2 was present in the air and on the surfaces of the ICU and GW (Ge et al. 2020). Passos et al. monitored two hospital facilities (indoor environment) and public spaces (outdoor environment) in Brazil from May to August 2020, and SARS-CoV-2 was found in aerosols inside the ICU, in the protective apparel removal room, in the room containing patient mobile toilets and used clothes (room with natural ventilation) and in an external corridor adjacent to the ICU (Passos et al. 2021).

Simulations of viral particle distribution within and outside of wards provide strong support for the prevention and control of hospital infection. In addition to the etiological characteristics of SARS-CoV-2, the transmission and diffusion rules of SARS-CoV-2 aerosols have been studied. A simulation of the spread of the virus conducted by scholars in Finland showed that the virus can spread instantaneously along an aisle when a COVID-19 patient coughs in a confined space in a supermarket (Vuorinen et al. 2020). The results showed that SARS-CoV-2 can be transmitted over a long distance and remain in the air for several minutes (Vuorinen et al. 2020). Li et al. evaluated the probability of a long-range aerosol spread of the SARS-CoV-2 having occurred in the poorly ventilated and crowded Restaurant X on January 24, 2020 by epidemiologic analysis, onsite experimental tracer measurements, and airflow simulations (Li et al., 2021b). Their findings suggested that it is crucial to prevent overcrowding and provide good ventilation and effective air distribution in buildings and transport cabins to prevent the spread of SARS-CoV-2 and the development of COVID-19 (Li et al., 2021b). Zhang et al. used the South China seafood market in Wuhan as an example in early 2020 to evaluate the risk of aerosol transmission using quantitative microbial risk assessment (QMRA) (Zhang et al. 2021). Based on the dose–response model, the key processes of viral deposition, diffusion, drift, decay, lung deposition and infection risk were integrated, and the number of COVID-19 beds available per capita in Wuhan (1.17 × 10^5) was taken as the manageable risk indicator. One hour after a customer contacted an infected shopkeeper in the market, the risk of infection with SARS-CoV-2 via the aerosol route was approximately 2.23 × 10^{-6} (95% C.I.: 1.90 × 10^{-6}–2.34 × 10^{-6}) (Zhang et al. 2021), which suggested that if multiple infected shop owners were attending the market at the same time, the risk could be increased several times, while the risk was reduced to <10^{-6} five meters away from the seafood market exit (Zhang et al. 2021).

However, studies on viral distribution and infection risk in negative-pressure wards are not sufficient. As hospitals are the main places to treat COVID-19 patients, infection risk assessment is crucial to protect healthcare workers and prevent the spread of the epidemic. Thus, this study aims to develop an infection risk assessment method to predict and manage the infection risk of SARS-CoV-2 in negative-pressure wards. The results of this study may provide a theoretical reference and technical support for reducing the occurrence of nosocomial infections.

2. Material and methods

2.1. Computational fluid dynamics modeling

2.1.1. Wuhan Huoshenshan Hospital ICU ward model

A physical model of the ICU in Wuhan Huoshenshan Hospital was created based on publicly available design information. The room measured \( L_x = L_y = L_z = 28.1 m \times 16.9m \times 3m \) (Fig. 1a); according to the actual situation of the ICU ward, 15 patient beds were evenly distributed in 15 cubicles, and the patients, medical staff, ICU bridges and office desks were distributed as shown in Fig. 1a. The personnel model was a 40-year-old Asian human solid model with a standing height of 1.750 m, a sitting height of 0.91 m and a body mass index (BMI) of 22 (Reed 2013). Three medical staff members were standing near a bed, two were sitting near a desk, two were in the buffer area, and one was in the pharmacy (Fig. 1a). A vertical view of the ICU ward is shown in Fig. 1b with details on room partitioning. Thirty-seven air inlets and sixteen air outlets were located in the ICU. The locations of the air inlets and the air outlets are shown in Fig. 1c.

2.1.2. Distribution of the particle size of human exhaled aerosols

Several studies have been conducted to investigate the size distribution and number of aerosol particles exhaled by humans. However, there is substantial variability in the results of these studies due to the use of multiple detection methods and to differences in the patients (Fabian et al. 2011; Holmgren et al. 2010; Papineni and Rosenthal 1997). Fabian et al. measured the number and size of aerosol particles exhaled by patients infected with influenza virus (Fabian et al. 2008). The geometric mean of the number of aerosol particles in the air exhaled by patients was 724; 87% of the aerosol particles were between 0.3 \( \mu m \) and < 1 \( \mu m \) in size, and 13% were between 1 \( \mu m \) and < 5 \( \mu m \) (Fabian et al. 2008). Because SARS-CoV-2 and influenza virus are single-strand envelope RNA viruses and share similar diameters of approximately 100 nm and the same transmission routes of aerosols and contact, the simulation of exhaled aerosols here is based on aerodynamic data from that study on influenza virus. In this study, the experimental results were used as the basis for the selection of six aerosol size classes, 0.3, 0.5, 0.7, 0.9, 1.1, and 3 \( \mu m \), and the number of exhaled aerosols was related to the size class. The moisture surrounding exhaled aerosol particles rapidly evaporates, drying the particles in less than a few seconds (Lenhart et al. 2004). Therefore, the particle sizes in this study were all postevaporation aerosol sizes, and aerosol particles in the evaporation process were not considered. Although the evaporated aerosol particles are small, they are still sufficiently large to carry thousands of pathogens and transmit disease.

2.1.3. Calculation settings

The computational fluid dynamics (CFD) package ANSYS 2021R1 was used in this study to simulate the airflow and particle dynamics in the ICU. The computational grid was generated with the Fluent-meshing tool using the polyhexcore method, resulting in 50 million polyhedrons. The solution was obtained with a pressure-based solver and a coupled method, which meets the requirements of computational accuracy and speed. The Euler–Lagrange method was used to simulate discrete relative aerosol particles by considering the effects of drag force, gravity, and Saffman force on particle motion. In practice, ICU wards are disinfected every 3 h, so the simulation time was chosen to be 3 h, and it was assumed that all
pathogens would be eliminated in each disinfection.

2.2. Calculation of viral concentration

2.2.1. Viral shedding in infected individuals

There are currently insufficient data on the relationship between individual aerosol-borne viruses and aerosol size. Ma et al. used reverse transcription-polymerase chain reaction (RT-PCR) to analyze SARS-CoV-2 viral concentrations in breath condensate samples collected from 49 patients, revealing values in exhaled gas of $10^7–10^9$ copies/m$^3$ based on the detected CT values (Ma et al., 2020). Assuming that all virions in the exhaled gas are attached to aerosol particles and that they can be modeled as a random variable obeying a log-normal distribution, the viral concentration in the aerosolized particles was calculated as:

$$C_{aerosol} = 10^{[\log_{10}(d_0) \times 10^{-12}] \times \frac{d_0}{6} \times V_T \times \sum N_i d_i^2}$$

where $C_{aerosol}$ is the number of virions contained per unit volume of aerosol particles, depicted in units of copies/m$^3$. $d_0$ and $d_T$ are the log mean and surface difference of the exhaled aerosol concentration, respectively; based on the data obtained by Ma et al. and assuming a normal distribution for the logarithm of the viral concentration in the exhaled gas, a log-normal fit was obtained, yielding values for $d_0$ and $d_T$ of 6.13 and 0.75, respectively. $d_0$ is the initial diameter of the exhaled aerosol, in mm$^3$; $N$ is the number of particles of the corresponding diameter exhaled; and $V_T$ is the tidal volume during a single breath, in m$^3$. The relation between the initial droplet diameter $d_0$ and the diameter of the equilibrium droplet nuclei $d_ae$ was estimated following the method proposed by Nicas et al. (Nicas et al. 2005):

$$d_{ae} = d_0 \times 0.16$$

Monte Carlo simulations were used to obtain $C_{aerosol}$ from 10,000 samples of data.

2.2.2. Comparison of experimental values

In a previous study, we sampled the viral concentrations on the surfaces of objects and in the air at different locations in the ICU ward of Wuhan Huoshenshan Hospital during COVID-19 patient admission, which provided actual viral concentrations to test the accuracy of the simulations (Guo et al. 2020). CFD simulations provided information about the location and timing of all aerosol particles in the ICU ward, and further processing of this information by using a computer program allowed the state of aerosol deposition on the surface of objects to be obtained. The number of virions deposited on the surface of different objects was calculated by the following equation:

$$C_{surface, test} = C_{aerosol} \times V_{aerosol, surface} \times A_{test}^{-1}$$

$C_{surface, test}$ is the viral concentration accumulated on the objects’ surface during the specified time, in copies/m$^3$; $V_{aerosol, surface}$ is the total volume of aerosol particles deposited within the sampling area of the object surface, in m$^3$, obtained by further processing of the computational fluid simulation results, which differ for different object surfaces at different locations; and $A_{test}$ is the sampling area, in m$^2$. In a previous study, we used cotton swabs to sample a fixed area of virus of approximately 5cm × 5cm on the object’s surface (Guo et al. 2020).

Airborne viruses were sampled by using a SASS 2300 wet wall cyclone sampler and collecting air samples at 300 L/min for 30 min (Guo et al. 2020). To realistically simulate the sampling process, a geometric model of the sampler was added to the research geometry model described previously, placed at the actual sampling location, and meshed and set with boundary conditions corresponding to the sampling process. A CFD model was created that could simulate the entire sampling process. The viral concentration in the air can be calculated by:

$$C_{air, test} = C_{aerosol} \times V_{aerosol, air} \times V_{air}^{-1}$$

$C_{air, test}$ is the concentration of virus accumulated on the surface of the object during the specified time, in copies/m$^3$; $V_{aerosol, air}$ is the total volume of collected aerosol particles, in m$^3$, obtained by further processing the computational fluid simulation results, which differ for different
2.3. Risk calculation

Virus-containing aerosols produced by patients while breathing are carried chiefly out of the vents with indoor ventilation airflow, partly deposited onto the surfaces of indoor objects, and partly dispersed in the air. Inhalation of virus-containing aerosols or contact with contaminated surfaces may cause transmission of SARS-CoV-2. In this study, the risk of infection due to close contact with the virus deposited on surfaces and the risk of infection due to inhalation of airborne virus-containing aerosols were calculated separately.

2.3.1. Exposure dose calculation for close contact

There is a lack of in-depth studies on the risk of infection through surface contact versus other transmission routes. Although some studies have concluded that the risk of infection through surface contact is very low (Wilson et al. 2021), we believe that more studies are needed to discuss the risk of surface infection in greater depth.

Viral transfer

Many factors affect the risk of infection caused by close contact, three of which are considered in this study: the area of contact, the half-life of the virus, and the transfer of the virus from the surface of an object to sites that may cause human infection, such as the eyes, mouth, and nasal cavity. A previous study by Wilson et al. concluded that the transfer of virus caused by contact with contaminated surfaces through the fingers is a function of the concentration gradient, and they used phage experiments and Bayesian simulations to estimate the surface transfer efficiency of virus from the object to the finger and from the finger to the surface of the mouth. Transfer parameter $\lambda_{\text{surface}}$ obeys a log-normal distribution: $\log_{10}(N) \sim N(-5.07, 0.11)$; transfer parameter $\lambda_{\text{mouth}}$ obeys a normal distribution: $\lambda_{\text{mouth}} \sim N(0.3390, 0.1)$, with the distribution truncated to 0 on the left and 1 on the right (Wilson et al. 2020). The frequency of hand contact with the human mouth, eyes, nose, and other parts of the body is specific to the activity and the individual. Mark Nicas and Daniel Best performed experimental statistics on 10 people for 3 h and found that the value can be taken as approximately 1 time/minute while performing common activities (Nicas and Best 2008). Viral biodecay

Studies have shown that SARS-CoV-2 has different half-lives on surfaces of different materials (van Doremalen et al. 2020). Based on research data, the half-life of SARS-CoV-2 varies widely from less than one hour to nearly 28 days (van Doremalen et al. 2020). In this study, four materials commonly used for objects in emergency medical buildings were selected: copper, cardboard, steel and plastic. The biological half-life of SARS-CoV-2 was considered a random variable obeying a normal distribution with truncated values on both sides as the maximum and minimum values obtained from the previous study as follows (van Doremalen et al. 2020): $T_{\text{copper}} \sim N(0.774, 0.195)$, $T_{\text{Cardboard}} \sim N(3.46, 0.81)$, $T_{\text{Steel}} \sim N(5.63, 0.65)$, $T_{\text{Plastic}} \sim N(6.81, 0.72)$. The number of active virions eventually transferred to the human mucosal surface was estimated by:

$$Dose_{\text{surface}} = \sum_{i} A_{\text{surface}} \times H \times \lambda_{\text{mouth}} \times \lambda_{\text{surface}} \times C_{\text{surface}}(t) \times dt$$

where $A_{\text{surface}}$ is the area of each hand-surface contact, the exact value of which depends on the mode of contact but was assumed to be 10 cm², which is approximately the surface area of 5 fingertips. $H$ is the hand-to-surface contact rate, and $t$ is the decay. $C_{\text{surface}}(t)$ is the viral concentration on the surface of the object, obtained by postprocessing the results of the CFD simulations through the program.

2.3.2. Exposure dose calculation of inhalation

Virions dispersed in the air can enter the respiratory tract by inhalation and cause infection. The process requires consideration of factors such as the intensity of human breathing and the deposition rate of aerosol particles in the respiratory system. In this study, the tidal volume of a human being in three typical conditions (sleep, sitting, and light exercise) was considered $V_s$, and the pulmonary ventilation volume was controlled by the following relation (Guha et al. 2014):

$$V_{E,sleep} = 28.042 \exp(0.0177b) N_{\text{resp}}$$
$$V_{E,sleep} = 12.774 \exp(0.0221b) N_{\text{resp}}$$

where $h$, the height of the individual, was taken as 175 cm based on the human modeling in this study, and the number of breaths in the human body $N_{\text{resp}}$ was taken as 20 breaths/minute.

In the ICU ward, the intensity of work of the medical staff can be primarily divided into moderate and vigorous activity (Ainsworth et al. 1993), with corresponding pulmonary ventilation volumes of 25.7 L/min and 44.3 L/min (Wang et al. 2017). To investigate the role of the protective equipment generally equipped by medical staff in hospitals admitting newly infected patients in reducing the risk of infection, we assumed that the medical staff may or may not be wearing N95 masks. The deposition rate of aerosol particles in the human body $f_{\text{mask}}$ was considered 95% with the mask and 0 without it (Qian et al. 1998).

The deposition rate of aerosol particles in the human body $f_{\text{mask}}$ was calculated by the pathogenic organism aerosol lung deposition model (Guha et al. 2014), in which the amount of virus deposited in the human respiratory tract by inhalation was calculated by the following equation:

$$Dose_{\text{breathing}} = V_{E} C_{\text{air}} f_{\text{mask}} (1 - f_{\text{mask}})$$

where $C_{\text{air}}$ is the viral concentration at the respiratory height and is obtained by postprocessing the results of the CFD simulations with the program.

2.3.3. Dose–response model

The relationship between exposure dose and risk of infection can usually be assessed with a dose–response relationship involving an exponential function. The exponential model is expressed as:

$$p = 1 - e^{-\frac{d}{k}}$$

where $p$ is the risk of infection at $Dose$, in copies, and $k$ is a pathogen-related parameter. Zhang Xiaole et al. derived the dose–response relationship based on the results of animal experiments, meta-analysis, etc. The constant $k$ for SARS-CoV-2 ranges from approximately $6.4 \times 10^4$ to $9.8 \times 10^5$ viral copies (Zhang and Wang 2021a). In this study, $k$ was assumed to obey a triangular distribution with lower, upper, and plural limits of $6.4 \times 10^4$, $9.8 \times 10^5$, and $1.6 \times 10^6$ copies, respectively.

The entire risk calculation process is simulated using Monte Carlo methods to fully account for uncertainties in various parameters.

2.3.4. Airborne infection risk calculation

The concentrations at the height of the medical staff members’ nose and mouth were selected to determine the relative infection risk from airborne exposure, considering that some medical staff are always standing at the headboard, the footboard, the corridor, and the pharmacy, while those in the office area are always sitting. The deposition of aerosol particles in the human respiratory system was estimated by using the International Commission on Radiological Protection (ICRP) pathogenic organism aerosol pulmonary deposition model. The Monte Carlo method was used to consider the uncertainty of viral shedding and the corresponding model constants for the dose. The infection risk calculated in this study was caused by aerosol inhalation within 3 h of breathing.

2.3.5. Surface infection risk calculation

The viral concentration on the surface of objects in the ICU ward increased as the aerosol particles are continuously deposited. The ICU
ward was disinfected once every 3 h, so the infection risk within 3 h was simulated and calculated. The gradient transfer model considers both the decay of virions on the object surface and the transfer between the object surface, the hands of the medical staff and the mucosa. The Monte Carlo method was used to calculate the infection risk through close contact with objects, taking into account the uncertainty of viral shedding, the surface half-lives of different materials, the object-to-hand concentration transfer rate, the hand-to-mucosal concentration transfer rate, and dose corresponding model constants.

3. Results

3.1. Viral concentration simulation in the ICU ward

In the ICU ward, viral particles are continuously exhaled by patients and carried by air partly out of the exhaust and partly diffused and gradually deposited throughout the ward as a dynamic process. The CFD results showed that it takes only 16 min for a completely clean ICU ward to enter a state in which the aerosol particles in the ward air are in dynamic equilibrium. This means that the concentration of aerosol particles in the air does not substantially change often. The aerosol concentration on the surface of objects in the ICU ward increased over time due to the cumulative nature of aerosol deposition.

3.1.1. Viral aerosol concentration in the ICU ward

The three-dimensional streamline diagram isosurface and the viral concentration isosurface of the ICU ward are shown in Fig. 2a and explain the formation of the high concentration region. The two air inlets of each cubicle entry are located over the footboard (Fig. 1b), and a strong counterclockwise return flow forms above the bed due to deflection by the sickbed (Fig. 2a). Exhaled aerosols are carried by the updraft of the return flow and then lifted and deflected in the direction of the air outlet. Most of the aerosol is carried to the air outlet by the deflected airflow after rising for a certain period of time (Fig. 2a); the rest continues to rise near the ceiling before being carried to the other side of the cubicle by the horizontal flow formed by the deflection from the ceiling. Based on the above flow trajectory, a high concentration area forms from the ceiling to the air outlet (Fig. 2b). A portion of the aerosol is then carried toward the floor by the vertical flow entering the air inlet on the bedside, and the vertical flow becomes horizontal due to deflection by the floor. The carried aerosol particles are partly deposited and partly passed through the bottom of

![Fig. 2. Results of simulated air flow and SARS-CoV-2 concentration in the ICU ward.](image)
the sickbed to the air outlet. Since most of the vertical airflow entering the air inlet at the end of the sickbed will be deflected near the floor and converge toward the air outlet, a small portion near the wall in the cubicle will be deflected toward the office area (A-A and B-B sectional plane in Fig. 2a). This deflected airflow will carry some of the aerosols in the direction of the office area, whose airflow will cause the aerosols to diffuse into the office area (Fig. 2a, b). Airflow interactions and obstructions from objects such as hospital beds and bridges can weaken the carrying capacity of the airflow, resulting in the deposition of some aerosols.

3.1.2. Cross-sectional distribution of viral aerosols in the ICU ward

Fig. 2b shows the distribution of viral aerosols in the ICU ward. The viral concentration shows a decreasing pattern from the cubicles on both sides to the office area (Fig. 2b). A funnel-like area of high viral concentration forms from the headboard to the ceiling of the cubicle, reaching 15.5 copies/L at its peak (Fig. 2b). However, most of the area has a concentration below 6.0 copies/L (Fig. 2b). At the headboard, footboard, corridor, and office area sections, viral concentrations first increase and then decrease with increasing height. The highest viral concentration at the headboard was found at 1.69 m with a concentration of 5.72 copies/L. The highest viral concentration at the footboard was found at 2.01 m with a concentration of 0.35 copies/L and at 1.70 m with a concentration of 0.197 copies/L. The viral concentrations in the corridor and office areas did not show much difference, ranging between 0.017 and 0.037 copies/L and 0.007 and 0.029 copies/L, respectively, with the highest concentration occurring at 1.75 m (A-A sectional plane in Fig. 2b). The viral concentration remained at approximately 0.017 copies/L at the height of the human mouth and nose in a sitting position (approximately 0.7–0.9 m). The viral concentration in the pharmacy did not show significant associations with height and ranged between 0.0035 and 0.0094 copies/L due to its distance from the beds and the barrier, with 0.0059 copies/L found at 1.70 m (B-B sectional plane in Fig. 2b). It was clear that a certain number of bioaerosol particles also entered the pharmacy with the airflow. Even based on the shortest migration path, the aerosol propagation distance exceeded 10 m, more than twice the 4 m estimated in our previous study (Guo et al. 2020).

3.1.3. Deposition of virions on the objects in the ICU ward

Fig. 2c shows the deposition of virions on the surfaces of the objects in the ICU ward at 3 h, demonstrating a greatly variable distribution of viral concentrations. The effect of the air outlets causes a large amount of airflow to accelerate toward them. The flow direction changes after encountering obstruction from the walls on both sides, and the particles dislocate to form an area of high concentration on the wall surface, in which the viral concentration was between $10^5$–$10^8$ copies/m$^3$ (B-B sectional plane in Fig. 2c). The strong vortex above the beds and the obstruction of the ICU bridge cause the particles to dislocate and stay, resulting in the formation of an area of high concentration on the surface of the ICU bridge and the head portion of the bed, with viral concentrations between $10^7$ and $10^8$ copies/m$^3$ at most locations on the ICU bridge surface and at the head part of the bed (B-B sectional plane in Fig. 2c). However, this does not mean that the viral concentration on all parts of the bed and ICU bridge is high; because of the complexity of the bed and ICU bridge structure, the viral concentration on individual parts, such as the gap in the upper part of the ICU bridge and the corner of the bed, is low or even close to 0 copies/m$^3$ (B-B sectional plane in Fig. 2c). The vertical downward airflow is deflected near the ground so that some of the particles are deposited in that direction. Additionally, a part of the horizontal airflow from the office area accelerates toward the air outlets; this part of the airflow has a higher particle carrying capacity, forming a high concentration area and a low concentration area on the floor in the cubicle, with the viral concentration in the high concentration area ranging from $10^4$ to $10^8$ copies/m$^2$ (A-A sectional plane in Fig. 2c). As part of the airflow coming from the air inlet over the end of the bed flows toward the office area, a zone of higher concentration extends toward the corridor. In the corridor area, the viral concentration is $10^4$–$10^5$ copies/m$^3$. The viral concentration in the office area is relatively low, and most of the desk surface is at $10^3$–$10^5$ copies/m$^3$ (A-A sectional plane in Fig. 2c). The viral concentration on the floor of the buffer area 1 is $0$–$10^4$ copies/m$^3$ (Fig. 2c). Notably, the floor of the pharmacy also contains virus due to natural aerosol deposition, with most of the area showing a concentration of $0$–$10^4$ copies/m$^2$ (Fig. 2c). The description above represents a typical flow for a relatively large part of the area, but a large range of viral concentrations in the ICU ward are influenced by the layout of the facility. For example, in the cubicles where the medical staff is placed, the distribution of viral deposition on the floor is very different from that of the other cubicles (Fig. 2c).

3.2. Data comparison between the simulation and field monitoring

In our previous study, the aerosol and surface distribution of SARS-CoV-2 in the ICU ward of Wuhan Huoshenshan Hospital were explored (Guo et al. 2020). The sampling was performed at different locations in the ICU ward, but it does not cover every possible situation. In this simulation, the viral concentration data in different parts of the ICU ward can be calculated.

3.2.1. Comparison of the simulated and monitored viral concentrations in the air

Data on the viral concentrations in the air were obtained by using an air aerosol particle sampler and the qPCR detection method. Because the sampler may affect the flow field during its sampling process, we established an ICU ward model containing the sampler model for the accurate simulation of the viral concentrations in the air according to the sampling parameters, such as flow rate and time.

Three sampling locations, sites 1, 2 and site 3, and the pharmacy are shown in Fig. 3a and Fig. 3b. The simulation results of the viral concentration at the pharmacy are shown in Fig. 3c; no virions were detected in the pharmacy, but the simulated median value was 0.017 copies/L. The simulation results and sampling data of site 1 are shown in Fig. 3d. Site 1 is located in front of the air outlet near the head of the bed. Here, the monitored value was 3.8 copies/L, and the median simulated value was 6.71 copies/L (95% CI: 5.81 to 8.14). At site 2, the monitored value was 1.4 copies/L, and the median simulated value was 3.95 copies/L (95% CI: 3.28 to 5.05). At site 3, the monitored value was 0.52 copies/L, and the median simulated value was 0.55 copies/L (95% CI: 0.36 to 1.01). Based on these data, the simulated viral concentration in the air is consistent with the monitored value.

3.2.2. Comparison of the simulated and monitored viral concentrations on the surfaces of objects

Surfaces in different parts of the ICU ward were sampled, including the sickbed handrails, cubicle floor, pharmacy floor and computer mouse in the office area (Fig. 4a). As shown in Fig. 4b, the monitored values for the pharmacy floor and the computer mouse in the office area were $2.8 \times 10^4$ copies/sample and $7.5 \times 10^4$ copies/sample, respectively, while the median simulated values were $3.06 \times 10^4$ copies/sample (95% CI: 5.92 \times 10^4 \text{ to } 1.16 \times 10^5) and $7.77 \times 10^4$ copies/sample, respectively (95% CI: 1.08 \times 10^5 \text{ to } 4.90 \times 10^5).

As shown in Fig. 4c, the viral concentration from the simulation of the sickbed handrails in each cubicle was quite different, with a median value of $3.34 \times 10^4$ copies/sample (95% CI: 3.10 \times 10^4 \text{ to } 3.66 \times 10^4), consistent with the monitored value of $4.3 \times 10^4$ copies/sample. The data in Fig. 4d show that the viral concentrations on the cubicle floor of beds 1–8 are smaller than those of beds 9–15, which may be due to ward asymmetry; the airflow in the middle of the ward flows more to the side with beds 1–8, reducing viral deposition in this part of the ICU. The median value from the simulation on the cubicle floor was $8.56 \times 10^2$. 

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copies/sample (95% CI: 7.97 × 10^2 to 9.33 × 10^2), which was much lower than the monitored value of 6.6 × 10^4 copies/sample.

3.3. Infection risk in the ICU ward

3.3.1. Infection risk from airborne exposure

The human infection risks under four different breathing states—moderate activity, vigorous activity, moderate activity with an N95 mask, and vigorous activity with an N95 mask—were calculated separately (Fig. 5a). The order of infection risk at different locations was as follows: headboard > footboard > corridor > office area > pharmacy. The infection risk was positively correlated with respiratory volume and viral concentration at different locations.

For example, while performing moderate activity, the infection risk for a medical staff member was highest at the headboard, with a median risk of 4.87 × 10^-2 (95% CI: 5.36 × 10^-1 to 2.43 × 10^-2). The median infection risk was 2.02 × 10^-3 (95% CI: 3.07 × 10^-2 to 9.86 × 10^-3) at the footboard, 2.64 × 10^-4 (95% CI: 4.07 × 10^-3 to 1.29 × 10^-3) in the corridor, 1.41 × 10^-4 (95% CI: 2.18 × 10^-3 to 6.89 × 10^-4) in the office area, and 6.04 × 10^-5 (95% CI: 9.30 × 10^-4 to 2.94 × 10^-3) in the pharmacy. These risks were approximately 19–20 times those during moderate activity with the N95 mask.

While performing vigorous activity, the median infection risk was 8.24 × 10^-2 (95% CI: 7.34 × 10^-1 to 4.18 × 10^-2) at the headboard, 3.48 × 10^-3 (95% CI: 5.23 × 10^-2 to 1.70 × 10^-4) at the footboard, 4.56 × 10^-4 (95% CI: 7.00 × 10^-3 to 2.22 × 10^-5) in the corridor, 2.44 × 10^-4 (95% CI: 3.75 × 10^-3 to 1.19 × 10^-5) in the office area and 1.04 × 10^-4 (95% CI: 1.60 × 10^-3 to 5.07 × 10^-6) in the pharmacy. These risks were approximately 19–20 times those during vigorous activity with an N95 mask. Fig. 5b shows the distribution of the risk of infection from airborne exposure at 3 h in different locations, showing that the distribution was concentrated mainly in the cubicles. Fig. 5c shows the risk distribution as a function of height in different locations. The highest risk at the headboard was at 1.69 m; at the footboard, 2.01 m; in the corridor, 1 m; in the office area, 1.7 m; and in the pharmacy, 1.5 m.

3.3.2. Infection risk of surface exposure

Fig. 6a shows the infection risk from surface exposure for four materials (copper, cardboard, iron, plastic) at five positions (ICU bridge, sickbed handrail, wall in the cubicle, floor beside the sickbed, and office desk). The infection risk of all the surfaces peaked at 3 h, with values in the following order: copper < cardboard < steel < plastic. When plastic
was selected for the facilities in the ward, the ICU bridge had the highest risk of all locations, at \(4.14 \times 10^{-7}\) (95% CI: 6.37 \(\times\) 10\(^{-1}\) to 1.72 \(\times\) 10\(^{-3}\)), followed by the sickbed handrail at \(4.57 \times 10^{-3}\) (95% CI: 1.03 \(\times\) 10\(^{-1}\) to 1.86 \(\times\) 10\(^{-3}\)), the wall in the cubicle at \(2.53 \times 10^{-3}\) (95% CI: 5.87 \(\times\) 10\(^{-2}\) to 1.03 \(\times\) 10\(^{-3}\)), the floor beside the sickbed at \(8.60 \times 10^{-9}\) (95% CI: 2.06 \(\times\) 10\(^{-3}\) to 3.52 \(\times\) 10\(^{-8}\)) and the office desk at \(1.10 \times 10^{-9}\) (95% CI: 2.61 \(\times\) 10\(^{-4}\) to 4.48 \(\times\) 10\(^{-7}\)). Fig. 6b shows a cloud map describing the infection risk of 3-hour plastic surface exposure; the risk distribution is self-evident.

### 3.4. Factors affecting the spread of SARS-CoV-2

#### 3.4.1. Infection risk under different ventilation conditions

During the operation of the ICU ward at Wuhan Huoshenshan Hospital, the ventilation system employs 12 air inlets and 24 air outlets per hour. We further calculated the infection risk from surface exposure and airborne exposure, assuming ventilation with four other conditions, including 12 air inlets and 12 air outlets, 12 air inlets and 16 air outlets, 16 air inlets and 24 air outlets, and 24 air inlets and 24 air outlets per hour.

Air changes per hour (ACHs) have a great impact on the infection risk from airborne exposure (Fig. 7a). For example, the calculation results for moderate activity indicate that 12 air inlets and 24 air outlets per hour may not be the best choice. The risk at the headboard is the highest, and the other four conditions show significantly different risk profiles from the standard 12 air inlet and 24 air outlet conditions. The ACH condition that resulted in the greatest reduction in the infection risk from airborne exposure was 16 air inlets and 24 air outlets, followed by 24 air inlets and 24 air outlets, 12 air inlets and 16 air outlets, 12 air inlets and 12 air outlets, and 12 air inlets and 24 air outlets. The airborne exposure risk for the 16 air inlets and 24 air outlets condition was at least 42 times lower than that at the headboard area and 20 times lower than that at the footboard area for the 12 air inlets and 24 air outlets condition.

Fig. 7b represents the infection risk from plastic surface exposure, a common material in the ward, at 3 h. In decreasing order, the highest median risk at 3 h was observed at the ICU bridge, followed by the sickbed handrail, wall in the cubicle, floor beside the sickbed, and office desk. The infection risk from surface exposure at the ICU bridge was 9 times higher than that of the sickbed handrail. However, the sickbed handrail is more frequently accessed by the medical staff and patients; the infection risk from the sickbed handrail was approximately 50 times that of the floor and 400 times that of the desk in the office area. In addition, the ACH had little effect on the infection risk of surface exposure; there was no significant difference in the effect of different ACHs at each position. These results may provide a reference for setting the ACH of emergency negative-pressure wards and ICU wards with similar structures in the future.

#### 3.4.2. Infection risk associated with SARS-CoV-2 variants

The viral load inhaled by COVID-19 patients is another critical factor that may affect the infection risk from surface and airborne exposure. Medical staff are usually performing moderate activities during patient care (bathing, dressing, and moving patients or physical therapy). When medical staff must contend with an emergency, they usually enter a vigorous activity state (using heavy nonpowered tools or moving items). An N95 mask is required to prevent aerosol infection. Fig. 8 shows the changes in infection risk from surface and airborne exposure, assuming that the viral copy number of a mutant strain is \(2^{7}\) times that of the common strain. Similar to previous figures, Fig. 8a shows the median simulated infection risk from airborne exposure as a function of viral load. As shown earlier, the order of infection risk is as follows: headboard \(\rightarrow\) footboard \(\rightarrow\) corridor \(\rightarrow\) office area \(\rightarrow\) pharmacy. The infection risk in the vigorous activity state was higher than that in the moderate activity state. For an increase in the viral load of \(2^{7}\) times, the median risk from airborne exposure during moderate activity at the headboard was approximately \(9.8 \times 10^{-3}\) (95% CI: 1 to 1.70 \(\times\) 10\(^{-1}\)). Equipping an N95 mask reduces the median risk to only \(1.78 \times 10^{-1}\) (95% CI: 9.99 \(\times\) 10\(^{-1}\) to 3.76 \(\times\) 10\(^{-2}\)). For an increase in the viral load of \(2^{7}\) times, the median infection risk of airborne exposure in vigorous activity at the headboard exceeds 0.96 (95% CI: 1 to 1.51 \(\times\) 10\(^{-1}\)). The use of N95 masks decreased the median risk to \(1.50 \times 10^{-1}\) (95% CI: 9.99 \(\times\) 10\(^{-1}\) to 3.76 \(\times\) 10\(^{-2}\)).
3.76 × 10^{-2}). For an increase in the viral load of 2^{10} (over 1000) times, the infection risk during moderate activity was approximately 7.1 × 10^{-1} (95% CI: 1 to 5.69 × 10^{-1}) at the headboard, 1.49 × 10^{-1} (95% CI: 9.66 × 10^{-2} to 7.63 × 10^{-2}) in the corridor, 8.29 × 10^{-2} (95% CI: 8.37 × 10^{-2} to 4.08 × 10^{-2}) in the office area, and 3.63 × 10^{-2} (95% CI: 5.41 × 10^{-2} to 1.74 × 10^{-2}) in the pharmacy. The main risk of infection remains concentrated in the cubicle. N95 masks can reduce the risk by nearly 20 times.

Fig. 8b shows the median infection risk from exposure to surfaces of four different materials as a function of viral load. Similar to previous results, the order of infection risk was ICU bridge > sickbed handrail > wall in the cubicle > floor beside the sickbed > office desk. The infection risk from all surfaces peaked at 3 h in the following order: copper < cardboard < steel < plastic. For an increase in the viral load of 2^{8} times,
the median infection risk at the plastic ICU bridge was approximately 1 (95% CI: 1 to 2.65 × 10^{-1}). For an increase in the viral load of 2 \times 10^2 (over 1000) times, the median infection risk was 9.41 \times 10^{-1} (95% CI: 1 to 1.54 \times 10^{-1}) for the steel sickbed handrail, 8.02 \times 10^{-1} (95% CI: 1 to 9.12 \times 10^{-2}) for the plastic cubicle wall, 5.39 \times 10^{-2} (95% CI: 6.98 \times 10^{-3} to 3.26 \times 10^{-3}) for the plastic sickbed floor, and 6.99 \times 10^{-3} (95% CI: 1.41 \times 10^{-4} to 4.13 \times 10^{-4}) for the mouse on the office desk. This indicates that the surface exposure risk can no longer be ignored. Our simulation results can provide reference data for determining disinfection intervals for the ICU ward.

4. Discussion

4.1. Quantifying pathogen exposure is a decisive step in quantitative risk assessment

Risk assessment is the basic procedure for controlling biosafety accidents. Qualitative analysis methods qualify and grade the probability of occurrence and the magnitude of consequences by relying on the knowledge, experience and intuition of the analyst, which requires the analyst to have sufficient experience and ability. Compared with qualitative assessment, quantitative risk assessment has increased accuracy and can provide more reference data for evaluating the infection risk from pathogen exposure. After the pathogen is identified, the
environmental exposure dose can be calculated by qualitative analysis of the exposure route, exposure intensity, exposure frequency and exposure duration using multiple methods, including direct detection, biomarker monitoring and model calculation. Haas et al. conducted a quantitative assessment of microbial risk in drinking water based on the dose–response model and found that the beta-Poisson model better described the probability of viral infection and estimated the risk of infection, clinical disease and death caused by viruses in environmental samples (Haas 2020).

In quantitative risk assessment, dose–response analysis can be used to estimate the direct relationship between pathogen exposure dose and infection probability. Currently, the most commonly used methods include epidemiological investigation and experimental data extrapolation. However, the implementation of epidemiological investigation methods is limited because of the difficulty in acquiring sufficient data. In experimental data extrapolation, a high-dose risk obtained from animal experiments is extrapolated to a low-dose risk in humans. Some examples of extrapolation models, which are more widely used than epidemiological investigations, include probit models, logit models, one-click models, multiclick models, multimorder models, log-normal models, and Webb models (Gehlhaus et al. 2011).

In this study, a visual flow field model was constructed to track the spatial distribution of viral aerosol particles in the ICU of Wuhan Huoshenshan Hospital (Fig. 1). Combining the characteristics of viral transmission by airborne or contact route and the law of viral survival and decay, the infection risk of different locations in the ICU was calculated, providing a reference for the risk assessment of nosocomial infection.

**4.2. Visualisation simulation of viral distribution can assist in the construction of monitoring and early warning systems**

Due to the special structure and operation and maintenance requirements of negative-pressure wards, real-time online monitoring and early warning of pathogenic microorganisms are necessary. Traditional bioaerosol monitoring and warning equipment relies mainly on prevention and control personnel to perform sample collection in specific places and wait for subsequent laboratory testing and analysis. However, the spatial distribution of viruses is very complex and variable (Fig. 2). Timely, effective, and comprehensive feedback on the required data is difficult with this kind of human-dependent bioaerosol monitoring. In addition, the purpose of environmental biohazard factors in monitoring is to perform early warning and elimination loop feedback. At present, there is an urgent need to develop an integrated environmental sample collection, monitoring, early warning and elimination system in high-risk sites.

CFD-based numerical modelling may play three important roles in monitoring and early warning systems. First of all, this model can predict the potential risk points and provide reference for the layout of monitoring sites according to the characteristics of the field environment and pathogens in the early stage of the construction of monitoring and early warning systems. Secondly, in the operation of the monitoring and early warning system, this model may predict the change of risk points in advance with the change of transmissibility caused by pathogen mutations, and guide the timely adjustment of field monitoring. Finally, this model may be used as a good backtracking method for pathogen infection, which can not only provide data for epidemiological investigation, but also further study the aerosol or surface transmissibility of pathogens in different environments, thus providing guidance for the formulation of protection strategies.

Automatic, intelligent and information-based bioaerosol monitoring and early warning technology has become an emerging trend. Hence, there is a need for alternative negative-pressure ward design monitoring and early warning networks that use the Internet of Things integrated with environmental aerosol monitoring technology. This would allow the identification of the main risks of unmanned monitoring and early warning systems, the display of all static and dynamic information from the equipment operational process of the monitoring and early warning management system in real time, and visualization of the three-dimensional management of big data during the whole life cycle of environmental leakage risk in negative-pressure wards, improving management efficiency and ensuring safe operation.

**4.3. Simulation of exposure risk can guide methods for improving nosocomial infection control measures**

A comparison of the surface inspection and simulation results revealed that the simulated values were consistently lower than the
actual monitored values (Fig. 3 and Fig. 4). There are a number of reasons for this observation. First, the diameter of the aerosol particles actually exhaled by the human body ranges from the submicron to the millimeter level and beyond, while the simulation only simulates the majority of particles, which measure below 10 µm. The simulation also ignores the patient’s coughing, sneezing, and speaking, which expel more aerosol particles than the normal breathing process. In addition, the activities of the medical staff and patients carry the virus to different locations through contact, which cannot be characterized by simulation. Among these factors, the first two basically have the same impact on the simulated values for different parts of the ward, while the third factor has a relatively large impact on the viral concentration in the ground near the sickbed, the pharmacy, and the office area. Due to the frequent movement of people in these areas, the soles of the shoes of medical personnel, clothing, etc., will carry the virus and lead to a greater concentration in these locations than the original aerosol concentration in the air.

Visual simulation of viral distribution can guide methods for improving nosocomial infection control measures. First, the risk of infection on the surface of different areas and objects in the ward is not consistent (Fig. 5 and Fig. 6), and the infection risk through airborne transmission may be altered by changing the ACH (Fig. 7), so the elimination cycle should be reasonably implemented depending on the location and materials. Second, the working hours of the medical staff
should be strictly controlled to strike a proper balance between individual work fatigue and infection risk. Because of the constant risk of infection in wards, some failures in biosecurity could lead to a sharp increase in infection risk; by increasing personnel training, the probability of error may be reduced. Moreover, it takes time to construct and apply the model, but this can be performed in advance to guide the construction of new negative-pressure wards. Finally, the variant strains demonstrated significantly increased viral loads and risks of infection (Fig. 8). The level of protection for nurses and surgeons should be increased when treating patients infected with variant strains, and new disinfection methods, electrostatic adsorption and other air purification methods should be added to all human environments.

This study provides more effective recommendations and scientific and practical guidance for reducing the risk of infection among health care workers in health care facilities that admit SARS-CoV-2-infected patients. Specifically, for negative-pressure wards, the simulations presented in this study suggest that the use of appropriate protective equipment such as masks is effective for reducing the infection risk from airborne exposure. Therefore, it is essential to provide health care workers with appropriate personal protective equipment such as N95 masks, face masks, and disposable gloves. In particular, when health care workers perform aerosol-generating procedures among suspected or confirmed COVID-19 patients, a particulate respirator should be worn. Ventilation has a significant impact on the risk from airborne exposure. A reasonable amount of ventilation can dilute the number of virus-laden aerosols in the air and improve the efficiency of aerosol removal. Reasonable ventilation settings are also important. Poor ventilation settings may result in aerosol particles not being exhausted in a timely manner or spreading to more distant locations, increasing the risk of infection. For example, when caring for or interacting with patients, it is best to avoid being on the side of the exhaust vent and to stay as far away from the patient’s head as possible. In addition, the infection risk of surface exposure should not be overlooked, so ward disinfection times need to be set appropriately to reduce the risk, with particular attention to surfaces such as drawbridges, beds, and certain confined areas. In contrast, environmental disinfection in the negative-pressure ward may require not only routine disinfection methods but also nucleic acid scavenging if conditions permit. Since the nucleic acid fragments of SARS-CoV-2 accumulated on the surface of the articles cannot be completely removed by each round of disinfectant, the subsequent monitoring results will be inaccurate.

4.4. Limitations and prospects of this study for addressing multiple issues

Above all, this study combines source term data from different studies. There are advantages and disadvantages to this approach of data combination. On the one hand, by integrating all authoritative data, we can restore the essence of things and build as comprehensive and practical a model as possible, especially a risk assessment model for COVID-19 infection in a specific environment. Some of these data come from the laboratory, and some of them come from clinical practices, which is difficult to acquire them in one team. On the other hand, there are still deficiencies in the existing data. For example, each reference study had its own specific environmental conditions and detection methods, and could not be unified in methodology. This is why we finally chose Huoshenshan as an important reference for the model prototype, because at least we have environmental monitoring data to serve as reference and evidence for model construction.

Besides, this study provides only the calculated data for negative-pressure wards, but many other countries and regions use nonnegative-pressure wards to treat patients. First, the original intention of this study was to investigate the law of SARS-CoV-2 transmission and infection risk in a specific space under multiple environmental factors. We chose the negative-pressure ward as the study scenario because we have sufficient on-site monitoring experience and data for this type of ward. Although this study did not calculate the SARS-CoV-2 distribution and infection risk in general wards, the visualization model can also be set by changing the boundary conditions to be applied to other scenarios in the future. Second, the practices of Wuhan Huoshenshan Hospital, which is the world’s first large-scale COVID-19 hospital, have been referred to by more countries and regions globally. For example, Chinese companies have helped three designated hospitals in Jakarta, the capital of Indonesia, build emergency negative-pressure isolation wards following the practices of Wuhan Huoshenshan Hospital, and 82 negative-pressure ward beds will be added to the three hospitals. There are tens of thousands of negative-pressure wards in operation or under construction around the world, and some common wards can also be modified to form a negative-pressure gradient. This study can provide not only a mathematical model for the calculation of nonnegative-pressure wards but also a reference for the prevention and control of hospital infection and the design of many negative-pressure wards in planning and construction. Finally, we hope that the mathematical model and calculation method presented in this study can be more widely discussed to calculate infection risk in other scenarios to provide data support for improving protective measures and preventing the spread of COVID-19.

Nevertheless, there are uncertainties that may affect the simulation results. First, the effect of personnel flow on the flow field and the variation in temperature and humidity in the ward were not considered in our model. Second, each patient was assumed to have the same and constant aerosol incidence in the model, and no consideration was given to the fact that coughing, sneezing, and other patient breathing processes increase viral release, which can fail to account for individual underlying biological and physical or behavioral differences. Third, we relied on a conventional discrete size cutoff to define aerosols, which should actually be continuous in size. Fourth, the complex movements of health care workers performing nursing and surgical tasks are difficult to simulate.

In addition, the impact of mutations on transmissibility and infectivity is polygenic and multifactorial. Viral shedding is one of the potential impact factors that may affect the high transmissibility of SARS-CoV-2 variants. Currently, B.1.617.2, a strain of B.1.617, is being continuously transmitted at a higher rate across the world. The latest findings from Public Health England (PHE) show that the B.1.617.2 variant is at least 40% to 50% more transmissible than the B.1.1.7 variant in the UK (Kirola 2021). B.1.617.2 is associated with significantly lower PCR Ct values and significantly longer duration of Ct values ≤ 30 (estimated median duration 18 days for B.1.617.2, 13 days for wild-type) (Ong et al., 2021). The association of B.1.617.2 with this lower Ct value and longer viral shedding provides a potential mechanism for its increased transmissibility. A study released by a team of researchers at the China Center for Disease Control in Guangdong Province noted that the relative viral loads of patients infected with the Delta variant were 1260 times higher than those with 2020 infections with clade 19A/19B viruses on the day when viruses were first detected (Li et al. 2021a). However, many other factors may also contribute to the transmissibility and infectivity of SARS-CoV-2, such as receptor binding and immune escape. This study only discusses differences based on the exhaled viral aerosols of patients infected with SARS-CoV-2 variants.

Furthermore, in this study, the aerosol transmission risk of SARS-CoV-2 in negative-pressure wards was quantitatively assessed by combining CFD simulations with a dose–response assessment framework and based on available information on SARS-CoV-2. The development of this modeling framework allowed us to move away from the uncertainty associated with the assumption of aerosol homogeneity in the room in traditional quantitative microbiological risk assessment (Azimi et al. 2021; Schijven et al. 2021; Zhang and Wang 2021b) and to extend the traditional CFD modeling of aerosol transport and dispersion to quantitative calculations of infection risk (Borro et al. 2021; Shrestha et al. 2021; Vernez et al. 2021). The probability of infection for medical personnel was estimated from two infection pathways: airborne exposure and surface exposure. The results of the study demonstrated that
Additionally, the increasing viral loads of SARS-CoV-2 variants may regularity and infection risk of SARS-CoV-2. The SARS-CoV-2 distribution data from this model were verified by field test data in the Wuhan Huoshenshan Hospital ICU ward. Based on this model, it was found that ACHs have a great impact on the infection risk from airborne exposure, while they have little effect on the infection risk from surface exposure. Additionally, the increasing viral loads of SARS-CoV-2 variants may potentially contribute to the varying infection risk. Therefore, this study may increase attention to how to improve nosocomial infection control measures. Further studies are needed to verify the knowledge of SARS-CoV-2 transmission proposed in this study.

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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Appendix A. Supplementary material

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