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**Role of pathogen-laden expiratory droplet dispersion and natural ventilation
explaining a COVID-19 outbreak in a coach bus**

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Abbreviations			
<i>ACH</i>	Air change rates per hour [h^{-1}]	<i>N</i>	Total released droplet number
ASHRAE	American Society of Heating, Refrigerating and Air-Conditioning Engineers	<i>n</i>	Number of droplets
<i>C</i>	Particle concentration [$\mu\text{g}/\text{m}^3$]	NMSE	Normalized mean square error
C_2H_6	Ethane	N_i	Passenger inhaled droplet number
C_c	Cunningham slip correction	N_s	Passenger inhaled pathogen-laden droplet number
CCTV	Closed-circuit television	<i>p</i>	Pulmonary ventilation rate [m^3/s]
C_d	Quantum concentration of droplets [quanta/ m^3]	<i>P</i>	Infection risk
C_e	Particle concentration at exhaust [$\mu\text{g}/\text{m}^3$]	PLD	Pathogen-laden droplets
CFD	Computational fluid dynamics	p_s	Pulmonary ventilation rate of index patient [m^3/s]
C_g	Tracer gas concentration	<i>q</i>	Quanta generation rate [quanta/s]
$C_{g,p}$	Tracer gas concentration at passenger's nose [ppm]	<i>Q</i>	Ventilation rate [m^3/s]
$C_{g,q}$	Quantum concentration of tracer gas [quanta/ m^3]	Re_p	Reynolds number
$C_{g,s}$	Tracer gas concentration at patient [ppm]	<i>RH</i>	Ambient relative humidity
$C_{i,s}$	Vapor concentration at droplet surface [$\text{kg}\cdot\text{mol}\cdot\text{m}^{-3}$]	RNG	Renormalization group
$C_{i,sr}$	Vapor concentration of bulk [$\text{kg}\cdot\text{mol}\cdot\text{m}^{-3}$]	SARS-CoV-2	Severe acute respiratory syndrome coronavirus 2
$C_{infected}$	Number of infected cases	$S_{susceptible}$	Number of susceptible people
COVID-19	Corona virus disease 2019	<i>t</i>	Time [s]
C_s	Particle concentration at inlet [$\mu\text{g}/\text{m}^3$]	<i>T</i>	Temperature [K]
C_v	Virus concentration	t_0	Exposure period [s]
<i>D</i>	Dilution ratio	T_e	Temperature at exhaust [K]
d_p	Initial droplet diameter [μm]	<i>TIF</i>	30-minute-exposure intake fraction
$F_{a,i}$	Additional forces [N]	<i>TIR</i>	30-minute-exposure infection risk
FB	Fractional bias	T_s	Temperature at inlet [K]
f_D	Stoke's drag modification	<i>U</i>	Normalized velocity
$F_{drag,i}$	Drag force [N]	$u_{p,i}$	Droplet velocity (m/s)
$F_{g,i}$	Gravitational force [N]	u_s	Supply air velocity [m/s]
<i>g</i>	Gravitational acceleration	<i>V</i>	Velocity [m/s]
<i>H</i>	Height [m]	V_{Bus}	Speed of bus [m/s]
HVAC	Heating, Ventilation and Air Conditioning	<i>Vol</i>	Volume [m^3]
<i>I</i>	Number of infectors	<i>W</i>	Width [m]
k_c	Mass transfer coefficient [m/s]	WHO	World Health Organization
<i>L</i>	Length [m]	<i>WIR</i>	Whole-journal-exposure infection
		$x_{p,i}$	Droplet displacement [m]
		<i>Z</i>	Poles height [m]
		μ_t	Turbulent viscosity [$\text{kg}\cdot\text{m}^{-1}\cdot\text{s}^{-1}$]
		ε	Normalized particle concentration
		θ	Normalized temperature
		λ	Molecular mean free path of air [m]
		ρ	Density of air [kg/m^3]
		ρ_p	Density of droplets [kg/m^3]
		τ_p	Aerosol characteristic response

Abstract

The influencing mechanism of droplet transmissions inside crowded and poorly ventilated buses on infection risks of respiratory diseases is still unclear. Based on experiments of one-infecting-seven COVID-19 outbreak with an index patient at bus rear, we conducted CFD simulations to investigate integrated effects of initial droplet diameters(tracer gas, $5\mu\text{m}$, $50\mu\text{m}$ and $100\mu\text{m}$), natural air change rates per hour($ACH=0.62$, 2.27 and 5.66h^{-1} related to bus speeds) and relative humidity($RH=35\%$ and 95%) on pathogen-laden droplet dispersion and infection risks. Outdoor pressure difference around bus surfaces introduces natural ventilation airflow entering from bus-rear skylight and leaving from the front one. When $ACH=0.62\text{h}^{-1}$ (idling state), the 30-minute-exposure infection risk(TIR) of tracer gas is 15.3% (bus rear) - 11.1% (bus front), and decreases to 3.1% (bus rear)- 1.3% (bus front) under $ACH=5.66\text{h}^{-1}$ (high bus speed).The TIR of large droplets(i.e., $100\mu\text{m}/50\mu\text{m}$) is almost independent of ACH , with a peak value($\sim 3.1\%$) near the index patient, because over $99.5\%/97.0\%$ of droplets deposit locally due to gravity. Moreover, $5\mu\text{m}$ droplets can disperse further with the increasing ventilation. However, TIR for $5\mu\text{m}$ droplets at $ACH=5.66\text{h}^{-1}$ stays relatively small for rear passengers(maximum 0.4%), and is even smaller in the bus middle and front($<0.1\%$). This study verifies that differing from general rooms, most $5\mu\text{m}$ droplets deposit on the route through the long-and-narrow bus space with large-area surfaces($L\sim 11.4\text{m}$). Therefore, tracer gas can only simulate fine droplet with little deposition but cannot replace $5\text{-}100\mu\text{m}$ droplet dispersion in coach buses.

Keywords: Computational fluid dynamics (CFD) simulation, droplet dispersion, infection risk (IR), natural air change rate (ACH), aerosol inhalation transmission, COVID-19

1. Introduction

Respiratory infectious diseases, such as influenza, severe acute respiratory syndrome (SARS) and coronavirus disease 2019 (COVID-19), have threatened public health in the last two decades [1]. In particular, the recent COVID-19 pandemic is caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). By 27 January 2022, more than 352 million people had been diagnosed with COVID-19, of whom more than 3.5 million had died[2]. Most of the human-to-human infections may occur in various indoor environments via droplet transmissions.

Numerous studies have demonstrated that respiratory infections may occur through the transmission of virus-laden droplets or droplet nuclei [3-5]. Pathogen-laden droplets (PLD) exhaled from the infected people may be the medium of human-to-human infection, because the pathogens can survive in the air for a period of time. Taking the current prevalence of COVID-19 for instance, the half-life of SARS-CoV-2 virus in the air is reported to be on the order of magnitude of one hour [6]. Recent studies have redefined that there are three main transmission routes of respiratory infection: surface touch transmission, drop spray transmission and aerosol inhalation transmission [7, 8]. Among them, aerosol inhalation transmission happens when air-suspended PLD are inhaled by a susceptible person. Ventilation modes and hourly air change rates (ACH) have been verified as the key factors affecting aerosol inhalation transmission and indoor infection risks [9-11].

There have been numerous studies on indoor ventilation, droplet/tracer gas dispersion and exposure analysis in inter-unit residential buildings [12-16], general indoor environments with mixing or displacement ventilation [17-19], and specific indoor space, for instance, restaurants [10, 20] and hospital wards [21, 22]. Particularly,

for the indoor environment of public transportation, most previous researches focused on airplane cabins [23-25] and high-speed trains [26, 27].

Although the coach bus is one of the most popular transportation modes for inter-city and suburban transportation, investigations on their indoor environments and infection risk are limited so far [28, 29]. Due to the unopenable windows, the crowded coach bus often obtains fresh air from the skylight suppliers which is related to the running conditions. When the HVAC (Heating, Ventilation and Air Conditioning) is on, there is only indoor circulation, resulting in an insufficiently ventilated indoor environment. Therefore, the coach bus is conducive to the transmission of respiratory infectious diseases, which is worthy of study.

Our previous researches [11] reported that there was a COVID-19 outbreak with 7 non-associated infected passengers on a coach bus with insufficient natural ventilation in January 2020 (winter) in Hunan Province, China. In this study, we first conducted field experiments, and then numerically simulated the tracer gas dispersion under the measured mean ventilation condition [11]. However, since the coach bus was driving in three states (high bus speed, low bus speed and idling), corresponding to different natural ventilation rates, a further detailed case study is required to investigate the influence of different natural ventilation rates on droplet dispersion and resulting infection risk. In addition, the initial diameter of exhaled droplets is in a large range (0.1-100 μm) [30, 31]. Moreover, numerous studies [18, 32, 33] have indicated that the droplet evaporation correlated to initial droplet diameters and RH significantly influence their gravity force and deposition effects, which integrates with turbulent diffusion, drag force, Saffman's lift force, and so on. Such complicated processes

should be considered for more comprehensive investigations on transmission mechanisms and assessments of human-to-human exposure and infection risk.

Many studies have confirmed that the transport behavior of droplets smaller than $5\text{ }\mu\text{m}$ is similar to that of tracer gas in a room [34-37]. However, the indoor environment of the bus is different from that of ordinary rooms, because the bus is long and narrow with more obstacles and more complex indoor airflow. In addition, RH is considered to influence droplet dispersion in indoor environments[17, 32, 38]. Therefore, two questions need to be answered: does tracer gas still have a similar transmission with droplets smaller than $5\text{ }\mu\text{m}$ in the bus environment? does RH still obviously affect droplet dispersion in the buses? For the bus environments, the feasibility of adopting tracer gas to replace fine droplets and the effect of RH on droplet dispersion need further exploration.

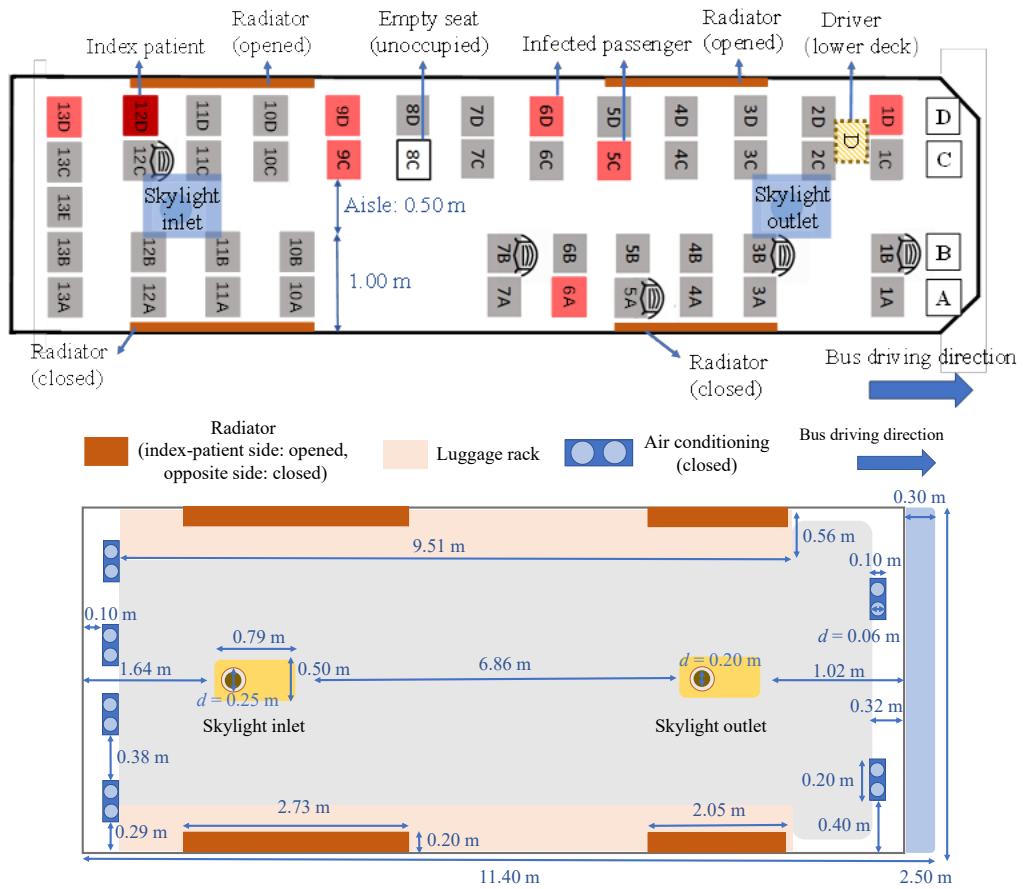
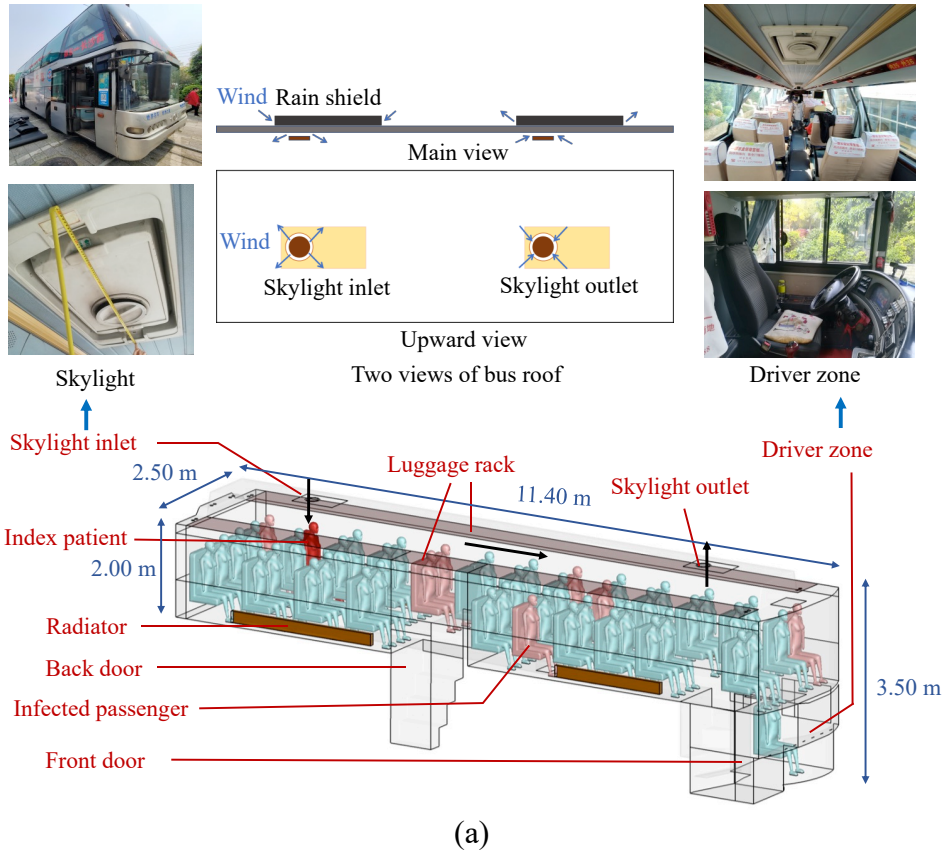
In the present study, by performing computational fluid dynamics (CFD) simulations, we further investigate the integrated impacts of RH (35%, 95%), initial droplet diameters (tracer gas, $5\text{ }\mu\text{m}$, $50\text{ }\mu\text{m}$, $100\text{ }\mu\text{m}$), natural ventilation rates ($ACH = 0.62\text{ h}^{-1}$, 2.27 h^{-1} , 5.66 h^{-1} respectively related to idling, low bus speed, and high bus speed), and body thermal plumes on the evaporation and dispersion of exhaled droplets in this enclosed coach bus. We also predict the difference of exposure/infection risk for various pathogen-laden expiratory droplets under different conditions.

2. Methodology

2.1. Full-scale experimental bus

[Fig. 1](#) shows the detailed model descriptions of the target coach bus with a cabin size of $11.4\text{ m} \times 2.5\text{ m} \times 2\text{ m}$ ($L \times W \times H$), where occurred a COVID-19 epidemic during the 200-minute long-route journey on January 22, 2020 [\[11\]](#). It was a double-deck 47-seat bus with the passenger cabin on the upper deck and the driver zone on the lower deck. Only the radiator at the index-patient side was functional and turned on during the whole journey, the other side was broken. As shown in [Fig. 1b](#), the cabin was fully occupied except Seat 8C (46 passengers in total). An index patient (in scarlet) at seat 12D returned home from Changsha city, Hunan province, China, eventually infecting seven of the passengers. These infected passengers (in pink) were respectively located at seats 1D, 5C, 6A, 6D, 9C, 9D and 13D. Among them, the passenger at seat 1D is farthest away from the index patient, at a distance of 9.46 m.

All the windows could not be opened with the skylights (as shown in [Fig. 1a](#)) for natural ventilation. Fresh air entered the bus through the skylight inlet at the rear ceiling, and contaminated air escaped from the skylight outlet at the front ceiling ([Fig. 1a](#)). The measured ACH could change with the various air pressure difference between the indoor and outdoor of the bus due to the various running speeds. More detailed information about the experimental setup can be found in our previous study [\[11\]](#).



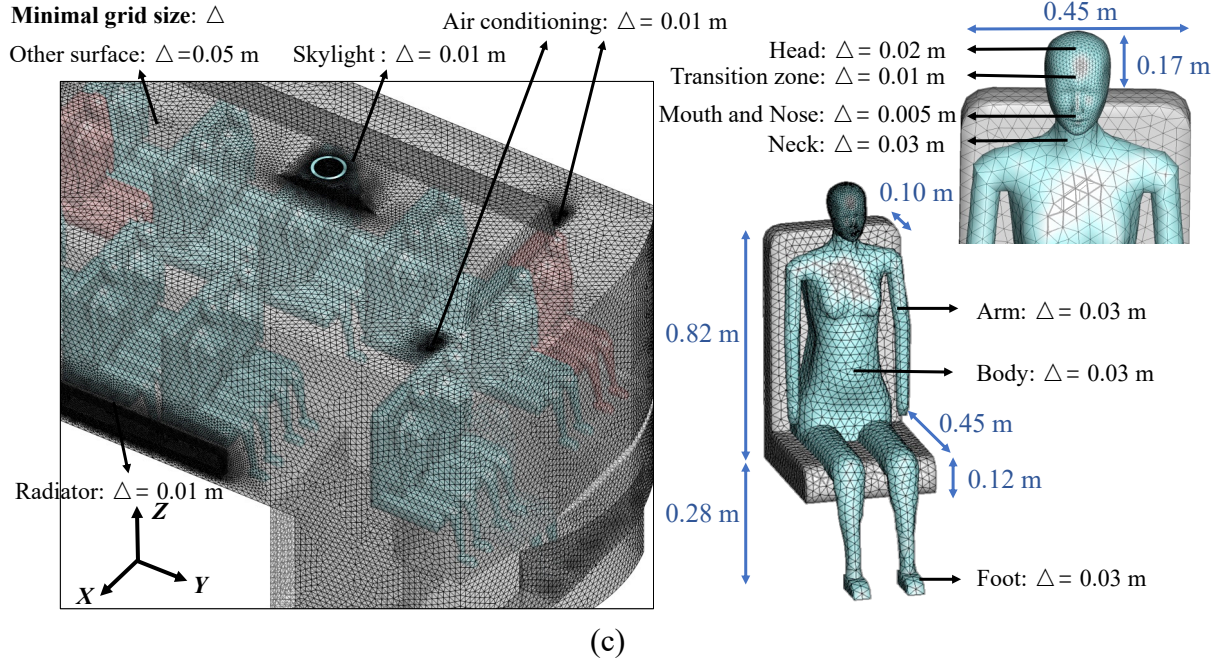


Fig. 1. (a) Bus photos and 3D bus model, (b) Bus top view and size information, (c) Grid arrangements of model.

2.2. Numerical modeling of bus ventilation and droplet dispersion

2.2.1. Descriptions of bus model and case studies

We utilized Gambit to build the bus cabin and manikin models (Fig. 1). We created a refined grid with 0.005 m size on mouth and nose, which is smaller than the grid of 0.03 m size around the human body, 0.01 m mesh size on the skylight inlet/outlet and heat radiator, and 0.05 m for the bus body (Fig. 1c). A total number of 5,379,993 unstructured meshes were generated, which was verified to ensure grid-independent requirements.

Twenty-one cases were considered as shown in Table 1. We investigated the influence of ACH (0.62, 2.27 and 5.66 h^{-1}) related to bus speed (0, 30 and 80-90 km/h), initial droplet diameters (tracer gas, $d_p = 5, 50$ and 100 μm), and ambient relative humidity ($RH = 35\%, 95\%$) on the transmission of the index patient's exhaled droplets

150 and the exposure/infection risk of other passengers. Ethane (C_2H_6) was adopted as the
 151 tracer gas to explore the dispersion difference between droplets and tracer gas in the
 152 coach bus.

153

Table 1

Parameters and setups in all 21 test cases.

Experimental variables	Setup	Notes
Ventilation rate (ACH / h^{-1})	0.62 h^{-1}	Bus is at idling ($V_{Bus} = 0$ km/h).
	2.27 h^{-1}	Bus is running at low speed ($V_{Bus} = 30$ km/h).
	5.66 h^{-1}	Bus is running at high speed ($V_{Bus} = 80-90$ km/h).
Initial droplet diameter ($d_p / \mu m$)	Tracer gas	As a surrogate for fine droplets and droplet nuclei.
	5 μm	For effect of initial diameter on droplet dispersion.
	50 μm	
	100 μm	
Ambient relative humidity ($RH / \%$)	35%	For effect of RH on droplet dispersion.
	95%	

154

155 **2.2.2. Numerical simulation of airflow model**

156 The renormalization group (RNG) $k-\epsilon$ model [39] has been verified to effectively
 157 simulate indoor airflows and tracer gas dispersion with considerable accuracy and
 158 computing efficiency [40-42]. Thus, we adopted Ansys FLUENT with RNG $k-\epsilon$ model
 159 to solve the conservation equations for mass, momentum, energy, humidity and
 160 turbulence variables. All the governing equations were discretized by the finite volume
 161 method in the second-order upwind scheme. SIMPLE scheme was selected to couple
 162 the pressure and velocity. Boussinesq hypothesis was adopted to consider the influence
 163 of thermal buoyancy.

164 To simulate the airflow field, we assumed that the variables were unchanging
 165 (steady) in the bus. CFD simulations were run until residuals became constant, for all

cases the iterations were over 100,000 times. Convergence was achieved after non-dimensional residuals for continuity equation, velocity components, energy, k and ε were below 10^{-3} , 10^{-4} , 10^{-6} , 10^{-4} and 10^{-4} , respectively and the monitored variables at specific surfaces were stable. We also checked energy balance and mass balance to help determine the convergence.

2.2.3. Droplet dispersion modeling

After the steady airflow field calculation was solved, we started the simulation of tracer gas dispersion and particle tracking, separately. The second-order upwind scheme was adopted in the tracer gas simulation. The mass fraction of C_2H_6 in the index patient's exhalation flows was 0.32 in CFD simulations according to [Ou et al. \[11\]](#). Lagrangian method with the Discrete Phase Modeling (DPM) was adopted to simulate the droplet dispersion with initial diameters of 5 μm , 50 μm and 100 μm [\[28, 43\]](#). Lagrangian equations of the droplets for i direction are as follows:

$$\frac{dx_{p,i}}{dt} = u_{p,i} \quad (1)$$

$$\frac{du_{p,i}}{dt} = \sum F_i = F_{drag,i} + F_{g,i} + F_{a,i} \quad (2)$$

$$F_{drag,i} = \frac{f_D}{\tau_p} (u_i - u_{p,i}) \quad (3)$$

$$F_{g,i} = \frac{g_i}{\rho_p} (\rho_p - \rho) \quad (4)$$

where $x_{p,i}$ and $u_{p,i}$ are the droplet displacement (m) and velocity (m/s) in i direction, respectively; $F_{drag,i}$ is the drag force ([Eq. \(3\)](#)), $F_{g,i}$ is the gravitational force ([Eq. \(4\)](#)). In addition, $F_{a,i}$ is the additional forces ([Eq. \(2\)](#)) for which we only considered Brownian force and Saffman's lift force [\[28, 44\]](#). ρ_p and ρ are the density of droplets and air,

respectively. f_D is the Stoke's drag modification function of Reynolds number for large aerosol (Re_p) [45].

$$f_D(Re_p) = 1 + 0.15 Re_p^{0.687} \quad (5)$$

In Eq. (3), τ_p is the aerosol characteristic response time, which is defined as:

$$\tau_p = \frac{\rho_p d_p^2 C_c}{18 \mu_t} \quad (6)$$

where μ_t is the turbulent viscosity ($\text{kg m}^{-1} \cdot \text{s}^{-1}$) and d_p is the droplet diameter. C_c is the Cunningham slip correction factor, which is defined as [46]:

$$C_c = 1 + \frac{2\lambda}{d_p} [1.257 + 0.4 \exp(-\frac{1.1 d_p}{2\lambda})] \quad (7)$$

where λ is the molecular mean free path of air.

In CFD simulations, the mass ratio of liquid (water) and solid element (sodium chloride) in droplets is assumed as 9 [47]. The densities of water liquid and sodium chloride are respectively 998.2 kg/m^3 and 2170 kg/m^3 . The evaporation process will continue until the droplets' volatile composition (i.e., water) is completely consumed. The vaporization rate is governed by the gradient of the vapor concentrations between the droplet surface and the bulk gas. The molar flux of vapor is defined as:

$$N_i = k_c (C_{i,s} - C_{i,sr}) \quad (8)$$

where k_c is the mass transfer coefficient (m/s) which can be obtained by Sherwood relationship [48]. The vapor concentrations at both droplet surface $C_{i,s}$ ($\text{kg} \cdot \text{mol} \cdot \text{m}^{-3}$) and bulk air $C_{i,sr}$ ($\text{kg} \cdot \text{mol} \cdot \text{m}^{-3}$) are calculated by the assumption of the ideal gas.

Table 2

Boundary condition setups in CFD simulation.

Boundary name	Boundary condition
Skylight inlet	Velocity inlet , velocity is different with various running conditions (idling: 0.57 m/s, low bus speed: 2.09 m/s, high bus speed: 5.37 m/s), temperature is 11 °C, turbulent intensity is 5 %.
Skylight outlet	Outflow , velocity is same as skylight inlet, temperature is different with various running conditions (idling: 24 °C, low bus speed: 23 °C, high bus speed: 21 °C), turbulent intensity is 5 %.
Glass at wall surface	No slip wall , heat flux is determined by energy balance estimates (idling: 92.89 W/m ² , low bus speed: 79.00 W/m ² , high bus speed: 56.63 W/m ²).
Index-patient side radiator	Standard wall function, no slip wall , heat flux is 100 W/m ² , surface area is 3.38 m ² .
Opposite side radiator (closed)	Standard wall function, no slip wall , heat flux is 0 W/m ² , surface area is 3.38 m ² .
Wall of bus, luggage rack, seat	Standard wall function, no slip wall , heat flux is 0 W/m ² .
Nose (except index patient)	Mass-flow-outlet , mass flow rate is 9.23×10^{-5} kg/s, Nostril area is 2.87×10^{-4} m ² .
Mouth of index patient	Velocity inlet , exhaled airflow velocity is 1.5 m/s (in a direction paralleling to Y-axis), temperature is 32 °C.
Other body surface	Standard wall function, no slip wall , convective heat is 50 W for each person.

207

208 [Table 2](#) shows the relevant boundary condition settings in CFD simulations [11].

209 At the skylight inlet, the ambient air temperature was set as 11 °C, and the inlet velocity

210 was set as 0.57, 2.09 and 5.37 m/s according to the measured *ACH*. At the skylight

211 outlet, the air temperature was set as 24, 23, 21 °C according to the experiment. Non-

212 slip wall with standard wall function was applied at all wall surfaces. The effects of

213 human respiration and body surface heat fluxes were considered, with convective heat

214 fluxes of 50 W for each sedentary passenger. To simplify the calculations, it was

215 assumed that the droplets were exhaled from the mouth of the index patient, while the

216 other passengers only inhaled through noses. A value of heat flux determined by the
 217 energy balance estimates was set on the bus window glasses [11].

218 After the steady airflow field with water vapor was solved, the single-diameter
 219 droplets were uniformly released from the mouth of the index patient at a rate of 20
 220 droplets per time step ($t = 0.1$ s, 18,000 iterations in total). The initial velocity of exhaled
 221 droplets was 1.5 m/s and the initial temperature was 32 °C. After 30 min continuous
 222 releasing, we got a fully-developed droplet distribution with a total droplet number of
 223 360,000. When a droplet encountered a surface, it would have three different fates: trap,
 224 reflect and escape. As shown in Table 3, different droplet sizes, surface roughness and
 225 other factors would lead to different boundary conditions of droplets on the surfaces
 226 [28, 49]. The trap condition was utilized for the floor, human surfaces and seats, which
 227 means droplets were trapped once they touched the objects and the trajectory
 228 calculations were terminated. While for the glass, roof, luggage racks and vertical walls,
 229 the reflect condition was applied due to smooth surfaces or gravity, which means
 230 droplets rebound off the surface and continue dispersion [28, 44, 50]. Escape condition
 231 was adopted to the skylight outlet and passengers' noses (except the index patient).
 232 Some of the CFD simulations in this study were completed on the Tianhe II
 233 supercomputer with the support of the National Supercomputer Center in Guangzhou.

Table 3

Boundary conditions of each boundary in Discrete Phase Model.

Boundary name	Boundary conditions
Skylight, nose of passenger (except index patient)	Escape (trajectory calculations terminated)
Glass at wall surface, roof, luggage rack, vertical wall	Reflect (droplets suspended in air)
Floor, air conditioning, radiator, human body surface, seat	Trap (trajectory calculations terminated)

234

2.3. Calculation of infection risk

We adopted the Wells-Riley equation to calculate each passenger's infection risk of aerosol inhalation transmission, which represented the probability of infection through inhaling PLD. This method has been verified to effectively predict the infection risk [51-53]. The Wells-Riley equation is defined as follows [54]:

$$P = \frac{C_{infected}}{S_{susceptible}} = 1 - e^{-\frac{Iqp}{Q}} = 1 - e^{-N_S} \quad (9)$$

where P is the probability of infection risk; $C_{infected}$ is the number of infected cases; $S_{susceptible}$ is the number of susceptible people; I is the number of people in the infectious stage or infectors; q is the quanta of PLD produced per infector per second (quanta/s); p is the pulmonary ventilation rate of each susceptible (m^3/s); Q is the room ventilation rate with virus-free air (m^3/s); t is the exposure time (s); N_S is the number of PLD inhaled by susceptible person, which was calculated for droplets and tracer gas by using different equations.

For droplets, N_S is defined as [54]:

$$N_s(x, t_0) = C_v p \int_0^{t_0} C_d(t) dt = C_v N_i \quad (10)$$

where C_v is the concentration of the virus in the exhaled droplets; $C_d(t)$ is the quanta concentration in an indoor environment at the time t (quanta/ m^3); t_0 is the exposure period; N_i is the total number of droplets (exhaled from the index patient) inhaled by passengers.

For tracer gas, N_S is defined as Eq. (11) according to the dilution-based evaluation method [55]:

$$N_s = \int_0^{t_0} p C_{g,q}(t) dt \quad (11)$$

where $C_{g,q}$ is the airborne quanta concentration at the target position (quanta/m³), and defined as $C_{g,q} = \frac{qC_{g,p}}{p_s C_{g,s}}$, where p_s is the breathing rate of the infector (m³/s); $C_{g,s}$ and $C_{g,p}$ are the airborne contaminant concentrations (ppm) at the source and target position, respectively.

Despite a critical parameter for calculating the infection risk, the value of q from a COVID-19 infector is currently not officially established. In this realistic bus outbreak, there were one infector ($I = 1$) and 45 susceptible passengers ($S_{\text{susceptible}} = 45$), seven of whom were infected ($C_{\text{infected}} = 7$). According to the travel history, this bus drove for 145 min at high speed ($Q = 5.69$ m³/min, $t = 145$ min), 45 min at low speed ($Q = 2.28$ m³/min, $t = 45$ min) and 10 min at idling ($Q = 0.62$ m³/min, $t = 10$ min). Substituting these data into Eq. (9) could back-calculate the value of q as 0.61 min⁻¹ (36.6 h⁻¹). This value agrees well with the range of 14–48 h⁻¹ obtained by Dai and Zhao [56] who adopted a reproductive number-based fitting approach.

Ansys FLUENT was employed to simulate the number of droplets inhaled by each passenger (N_i) and the concentration of tracer gas ($C_{g,s}$ and $C_{g,p}$). Then the N_s for droplets and tracer gas were respectively obtained by Eq. (10) and Eq. (11). Finally, the infection risk of each passenger was calculated by Eq. (9) for both droplets and tracer gas. In order to compare the infection risk under various ventilation rates, we carried out a 30-minute simulation for each vehicle driving situation.

2.4. Validation of numerical modeling

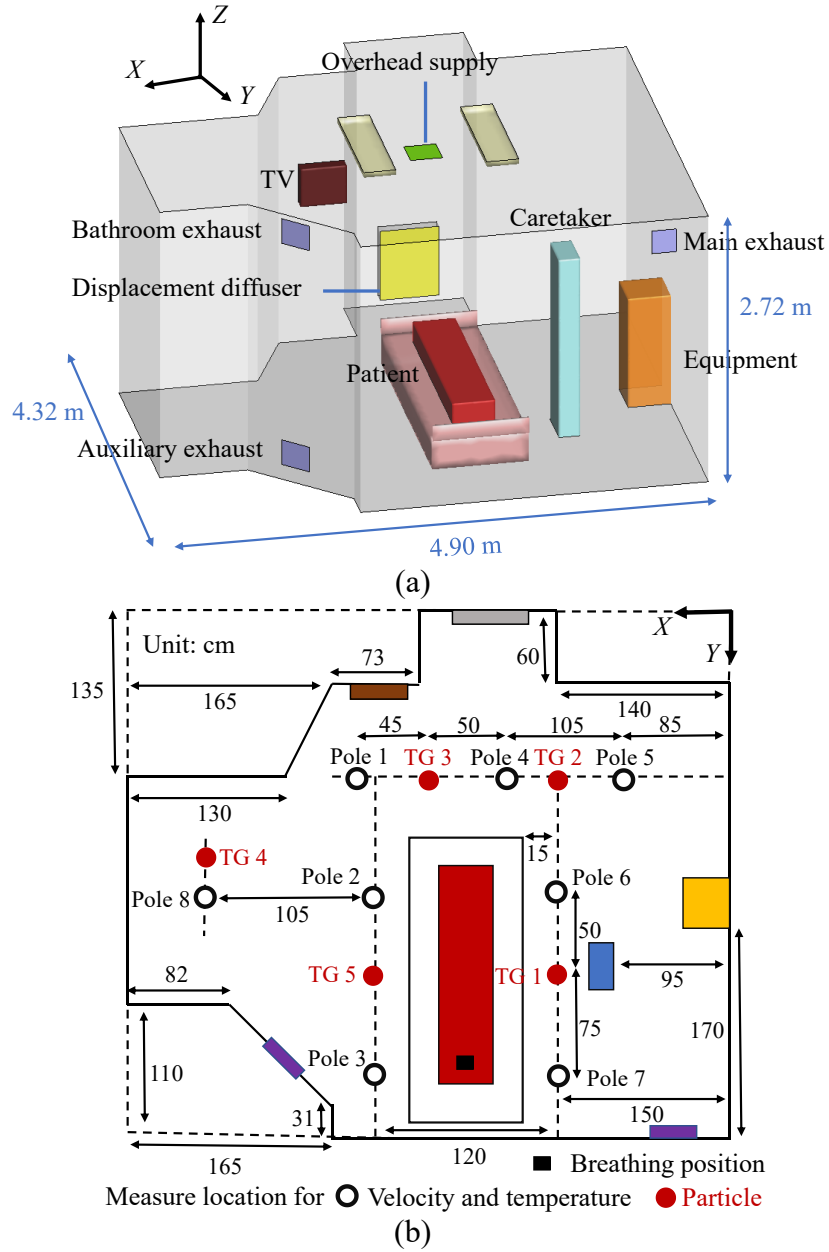


Fig. 2. Model setups of single-bed isolation room in CFD validation case (Yin et al., 2009).

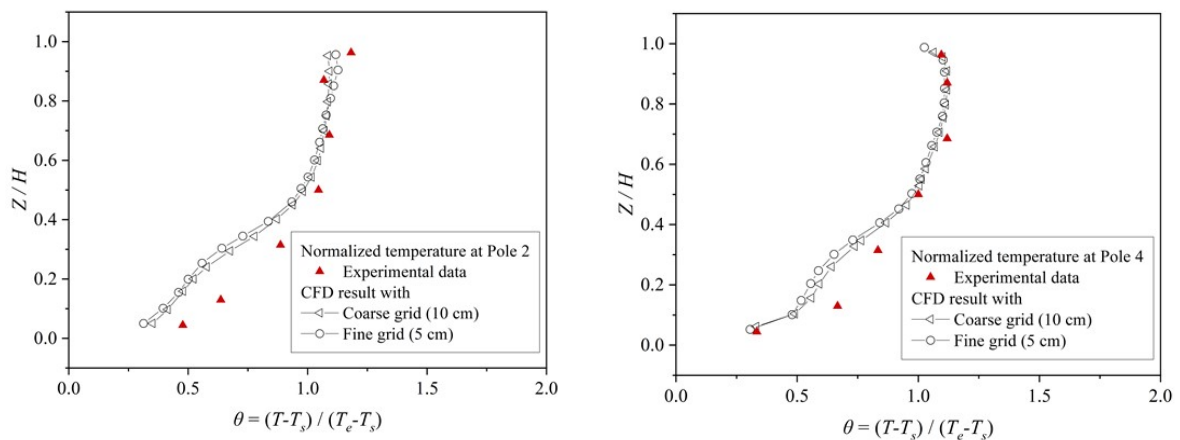
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280 Firstly, we performed a set of dispersion tests for validating the CFD predictions
 281 of tracer gas dispersion. The evaluation of tracer gas dispersion by this field
 282 experimental data can refer to our previous study [11].

283 In addition, we further carried out a validation study of indoor airflows,
 284 temperature and tracer gas/particle dispersion in a hospital ward evaluated by the
 285 experiment conducted by Yin et al. [36]. As shown in Fig. 2a, the experiment was
 286 performed in a full-scale one-person patient ward (4.90 m × 4.32 m × 2.72 m). The
 287 ventilation rate from the displacement diffuser was 0.054 m³/s (4 ACH) and the
 288 temperature was 19.5 °C. The ventilation rates of bathroom exhaust and main exhaust
 289 were 0.017 m³/s and 0.037 m³/s, respectively. As shown in Fig. 2b, the air velocity and
 290 temperature were measured at seven heights of Poles 1-8. Particle concentration was
 291 measured at poles TG1 - TG5 at six heights. More details about the experimental setups
 292 could be found in Yin et al. [36].

293 In this validation, we adopted 1.8 million and 3.8 million tetrahedral grid cells with
 294 fine and coarse grid resolutions, respectively. Then, we selected the measured vertical
 295 profiles of normalized velocity (V/u_s , $u_s = 0.14$ m/s means the supply air velocity) and
 296 temperature ($\theta = (T - T_s)/(T_e - T_s)$) at Pole 2 and Pole 4 to validate CFD results, where T_s
 297 and T_e are the temperatures respectively at diffuser and main exhaust at the normalized
 298 height (Z/H , $H = 2.72$ m is the height of the inpatient ward).

299



(a)

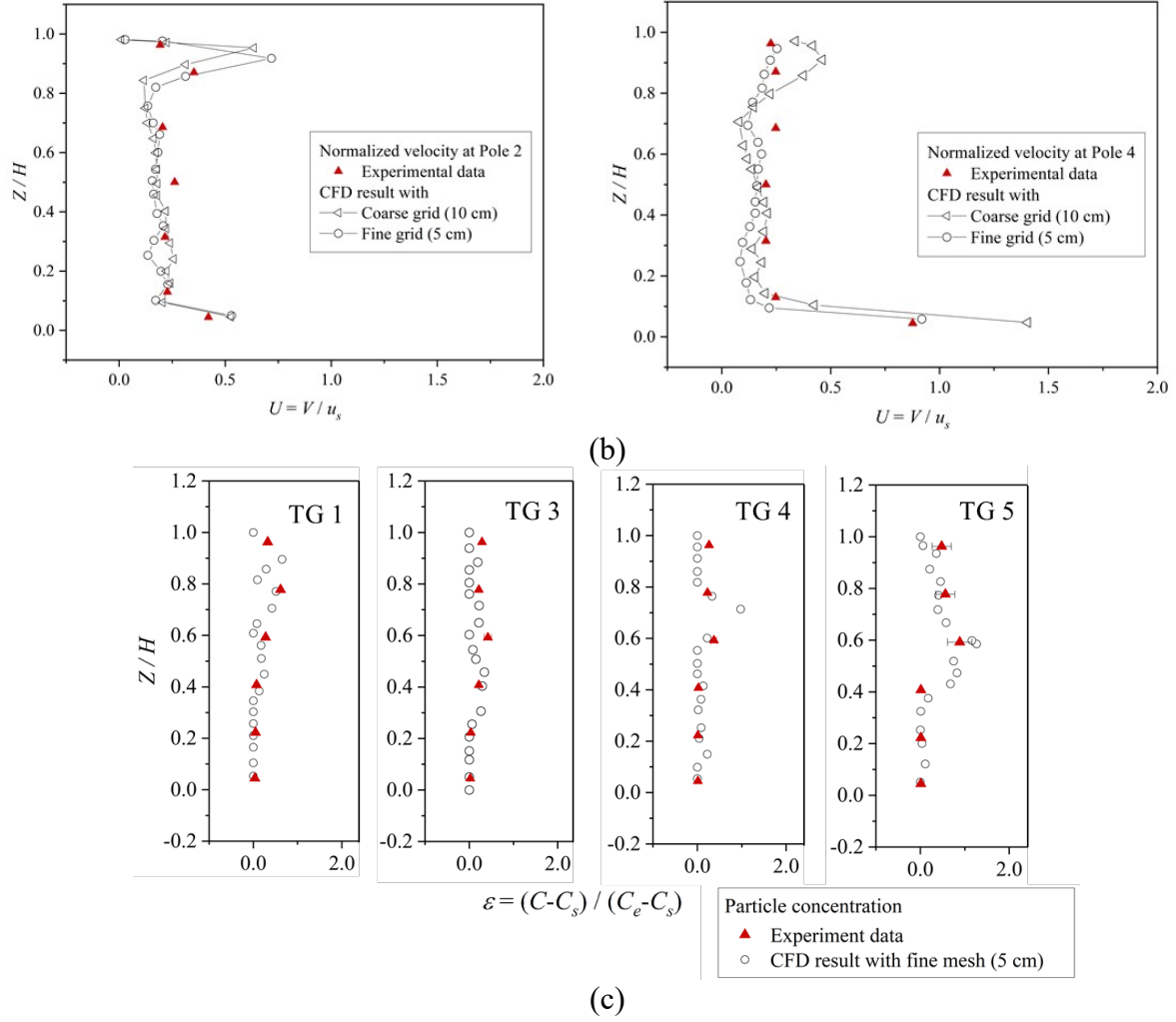


Fig. 3. Vertical profiles of experiment and CFD simulation at Pole 2 and Pole 4 (a) Normalized temperature, (b) Normalized velocity; (c) Normalized 1 μm particle concentration at TG 1, TG 3, TG 4 and TG 5.

300

301 [Fig. 3a-b](#) illustrate that CFD results of velocity and temperature agree well with
 302 the experimental data. Compared with the coarse grid resolution, fine grid resolution
 303 performs better, especially in the velocity at height of $Z/H = 0.8$. [Fig. 3c-f](#) display the
 304 experimental data and CFD simulation results of normalized particle concentration ε at
 305 TG1, TG3, TG4 and TG5 ($\varepsilon = (C - C_s) / (C_e - C_s)$, where C , C_s and C_e are the particle
 306 concentrations at the measuring location, ventilation supply inlet and ventilation
 307 exhausts, respectively). Particles with a diameter of 1 μm are released from the patient's

mouth. In order to quantify the reliability of the validation, we calculated the normalized mean square error (NMSE) and fractional bias (FB), whose ranges were respectively 0.3 to 1.3 and 0.01 to 0.27, which satisfied the recommended criteria (($NMSE \leq 1.5$, $0.3 \leq FB \leq 0.3$) in Yang et al [57]. The results indicate that CFD simulation results can reasonably predict the dispersion tendency of indoor particles.

3. Results

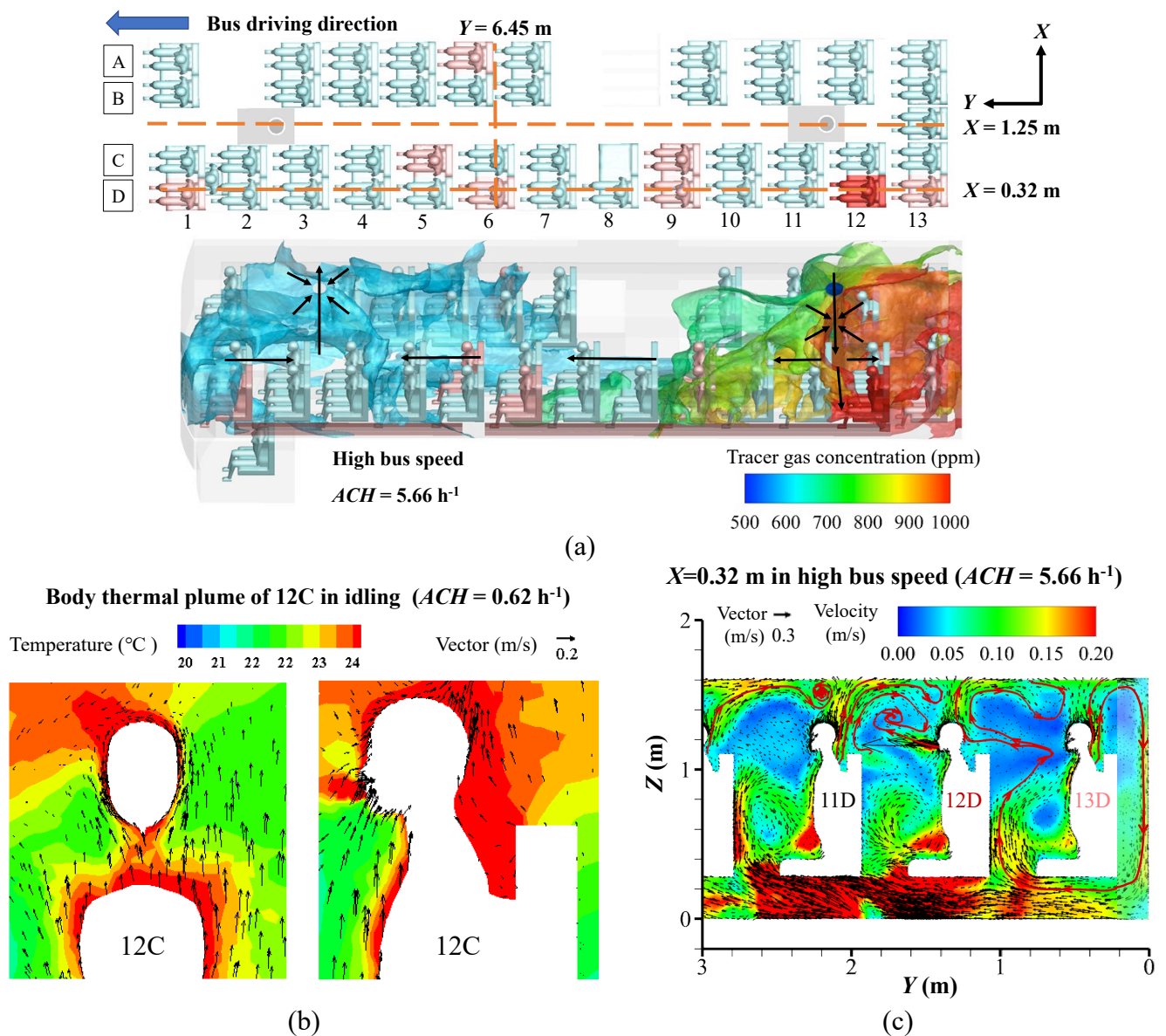
3.1. Flow pattern and tracer gas dispersion under various ACH

Our previous study [11] only considered the tracer gas dispersion under the measured mean ventilation condition. In the present study, we emphasize the flow pattern and tracer gas dispersion under different ventilation conditions (i.e., $ACH = 0.62$, 2.27 and 5.66 h^{-1}).

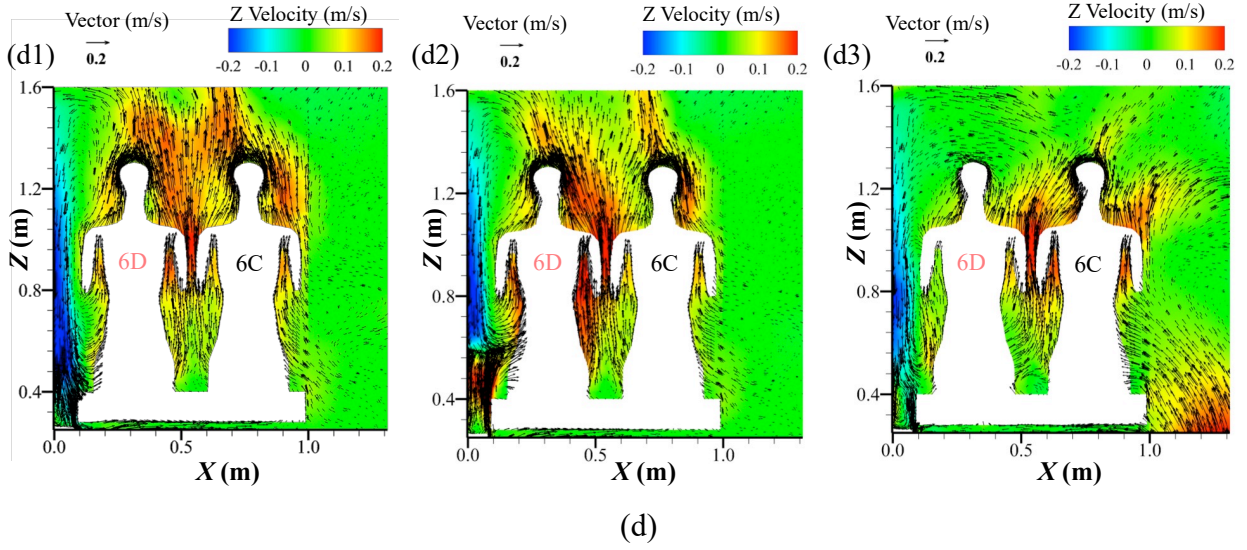
In order to describe the flow pattern and tracer gas dispersion more clearly, seat locations are shown in Fig. 4a: 13 rows (Row 1-13) and 5 columns (Column A, B, C, D, E) of seats in the passenger cabin. We regard the 1st to 4th rows as the bus front, the 5th and 8th rows as the bus middle and the 9th to 13th rows as the bus rear. The index patient is located at seat 12D (Row 12, Column D, scarlet).

Fig. 4a indicates that when the bus speed is high ($ACH = 5.66 \text{ h}^{-1}$), the fresh air enters the skylight inlet at the rear roof, then mixes with the dirty air and moves from the rear to the front. Finally, the mixed air leaves through the outlet at the front roof of the bus. There are body thermal plumes which lead to significant upward airflow near and above human bodies (Fig. 4b). The upward airflow will intertwine with the main flow field and subsequently affect the droplet dispersion. Fig. 4c displays that the

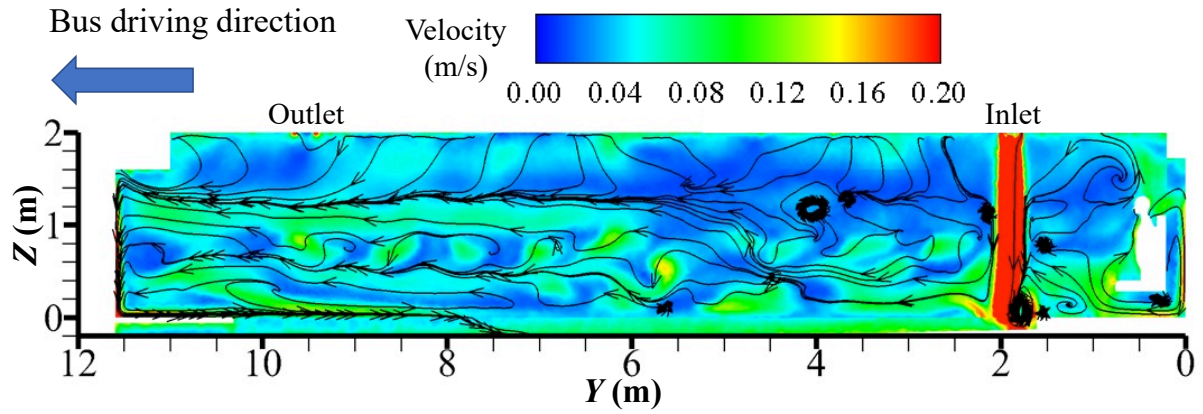
331 airflow exhaled by the index patient first moves forward, then rises up and finally
 332 deflects backward to the bus rear. As depicted in Fig. 4d, the body thermal plumes are
 333 most obvious under idling condition ($ACH = 0.62 \text{ h}^{-1}$). The ventilation flow from bus
 334 rear to bus front enhances significantly as ACH rises with the increasing bus speed (Fig.
 335 4e).
 336



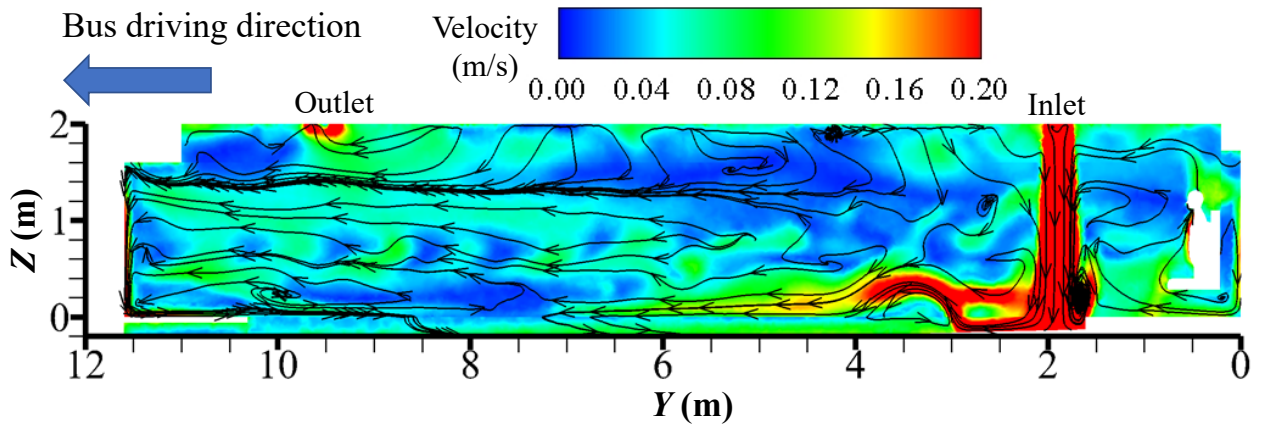
$Y=6.45$ m in idling ($ACH=0.62$ h $^{-1}$) $Y=6.45$ m in low bus speed ($ACH=2.27$ h $^{-1}$) $Y=6.45$ m in high bus speed ($ACH=5.66$ h $^{-1}$)



(e1) $X = 1.25$ m in idling ($V_{Bus} = 0$ km/h, $ACH = 0.62$ h $^{-1}$)



(e2) $X = 1.25$ m in low bus speed ($V_{Bus} = 30$ km/h, $ACH = 2.27$ h $^{-1}$)



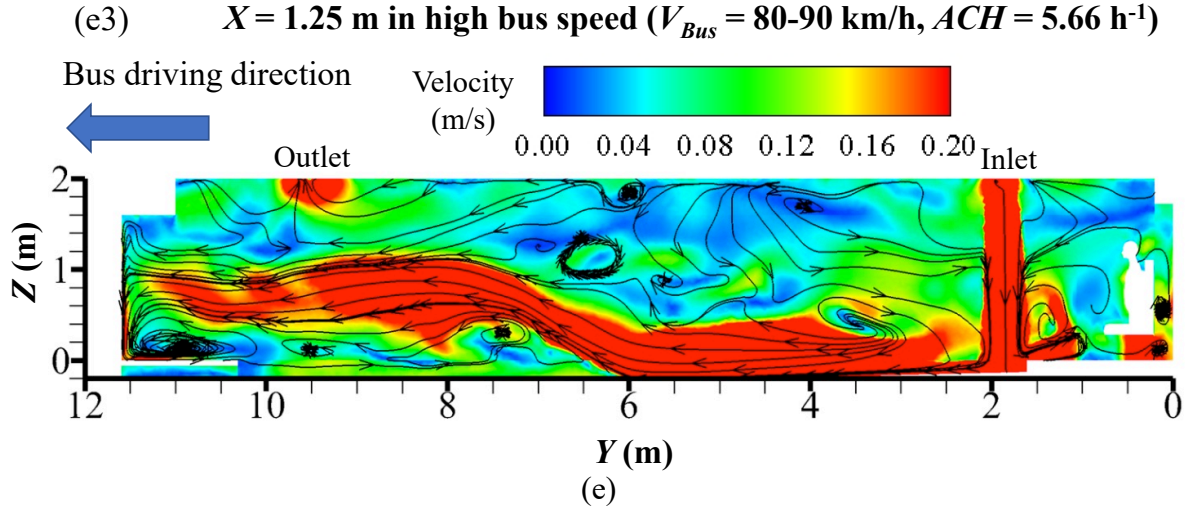


Fig. 4. (a) Cross-section line and general flow characteristics; (b) Body thermal plume of 12C at state of idling; (c) Flow field near index patient at state of high bus speed; Flow field in Plane: (d) $Y = 1.35$ m; (e) $X = 1.25$ m. 1-3: idling, low bus speed, high bus speed, respectively.

337

338 [Fig. 5](#) presents the tracer gas concentration distribution of the cross-section at the
 339 height of passengers' noses ($Z = 1.16$ m) under different ACH . Tracer gas concentration
 340 in the bus decreases obviously when ACH increases. High tracer gas concentration
 341 mainly appears in the bus rear (Rows 9-13), especially at the index patient's side
 342 (Columns C and D). Particularly when $ACH = 0.62$ h⁻¹, the concentration near the index
 343 patient is about 7000 ppm, and 5000 ppm in the bus front and middle (Rows 1-8) ([Fig.](#)
 344 [5a](#)). When $ACH = 5.66$ h⁻¹, the concentration near the index patient is about 1200 ppm,
 345 while around 600 ppm in the bus front and middle. ([Fig. 5c](#)).

346

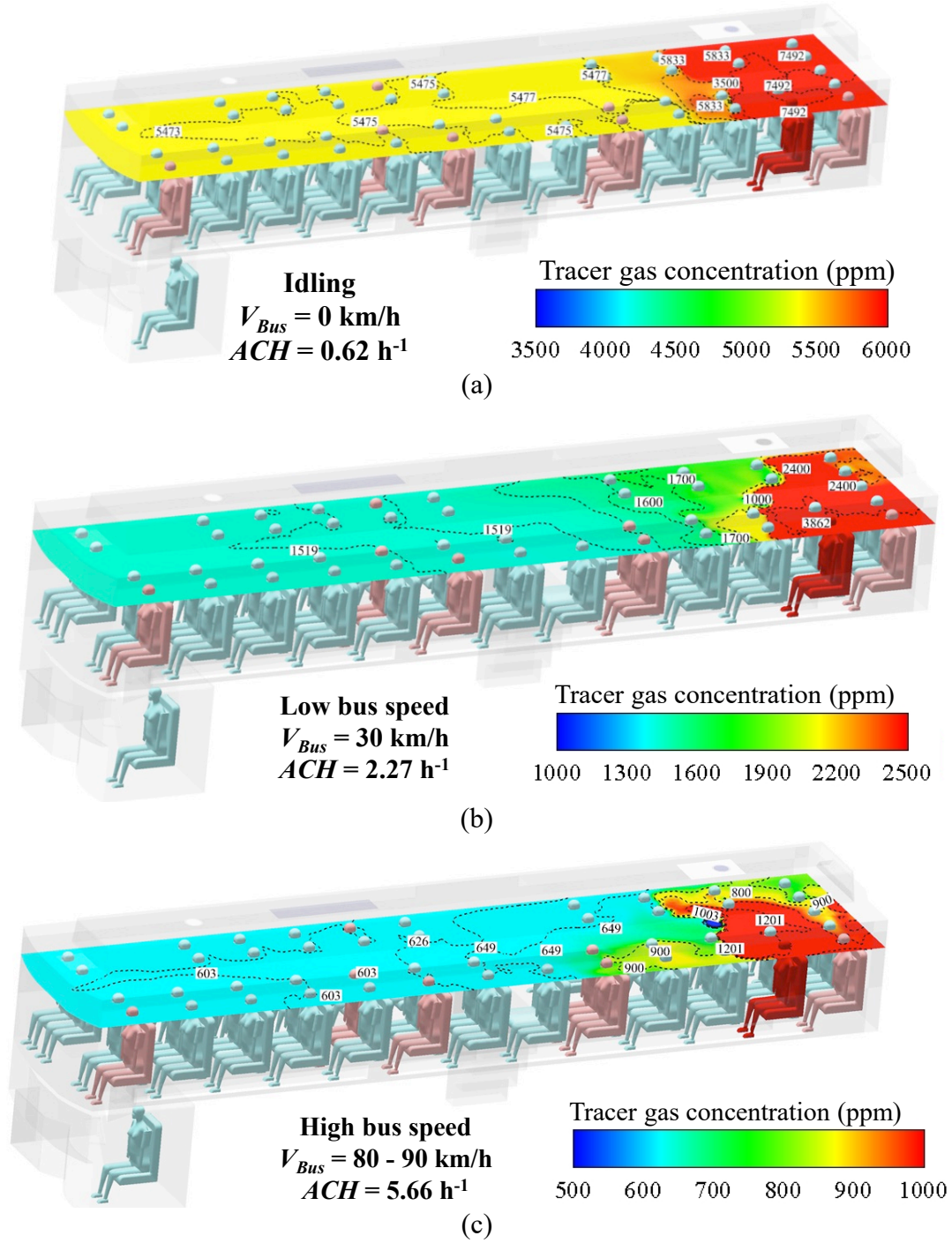


Fig. 5. Tracer gas concentration at state of: (a) idling, (b) low bus speed; (c) high bus speed.

3.2. Impacts of RH, ACH and initial diameters on droplet dispersion

3.2.1. Impacts of RH on droplet evaporation and transmission

Fig. 6 displays the temporal variation of droplet diameters (droplet evaporation history) under different RH (35%, 95%) when initial diameter d_p is 50 μm and 100 μm . When $d_p = 5 \mu\text{m}$, droplets can evaporate into 1.83 μm nuclei within 0.1 s under both RH conditions, so we don't show its evaporation process here. The results confirm that 50 μm droplets can evaporate into 18.26 μm droplet nuclei in 1.3 s under RH = 35% and 2.1 s under RH = 95%. However, it takes 5 s for 100 μm droplets to evaporate into 36.50 μm nuclei under RH = 35% and longer time (> 6 s) under RH = 95%. Although droplets take a longer time to evaporate in more humid environment, the overall impact of RH is not significant in this coach bus with complicated interactions of ventilation airflow and thermal body plumes (Fig. S1).

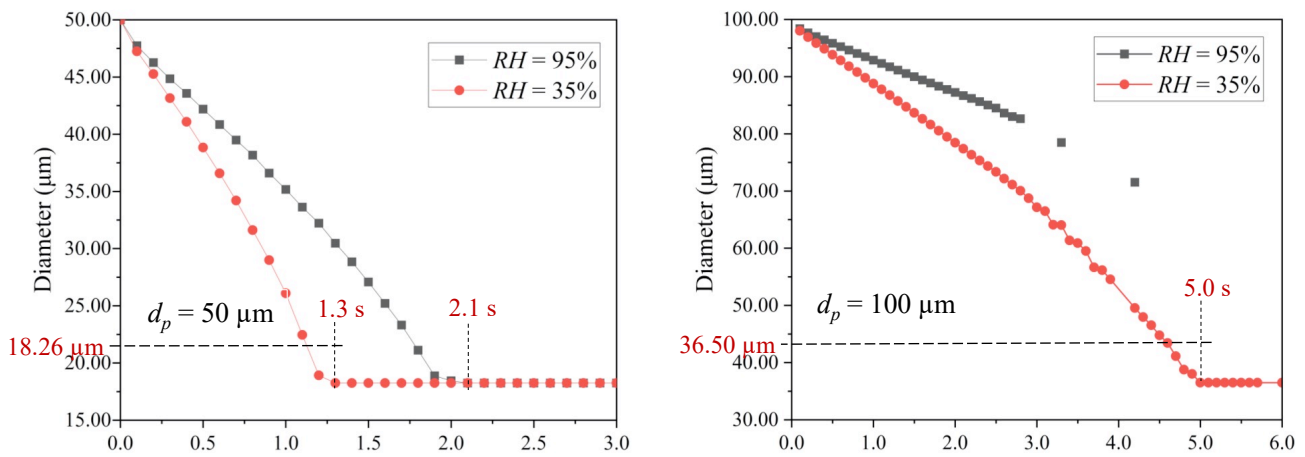
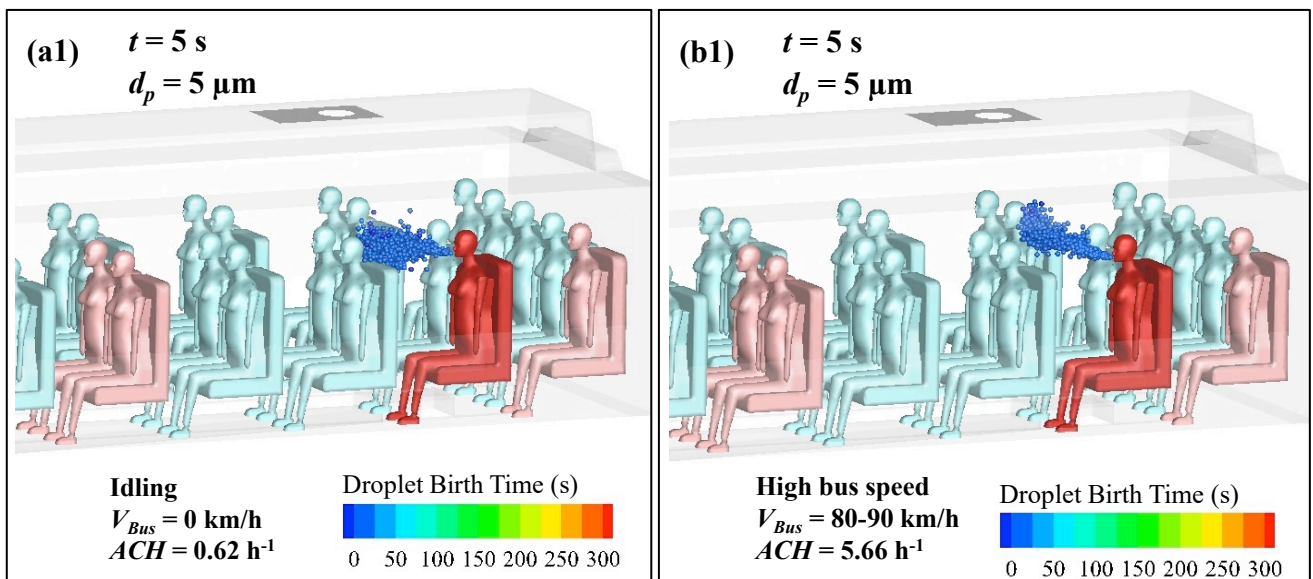


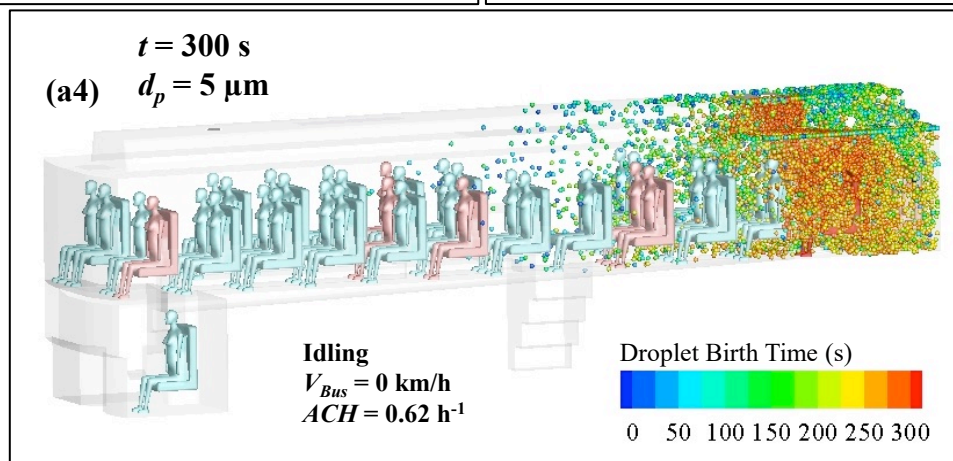
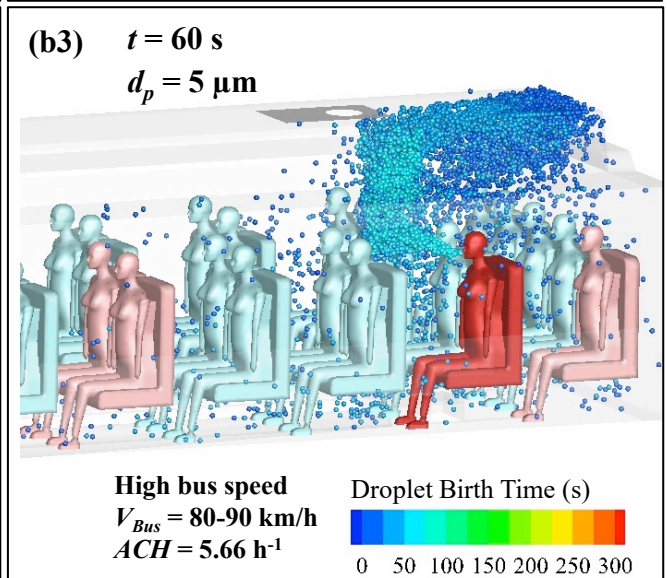
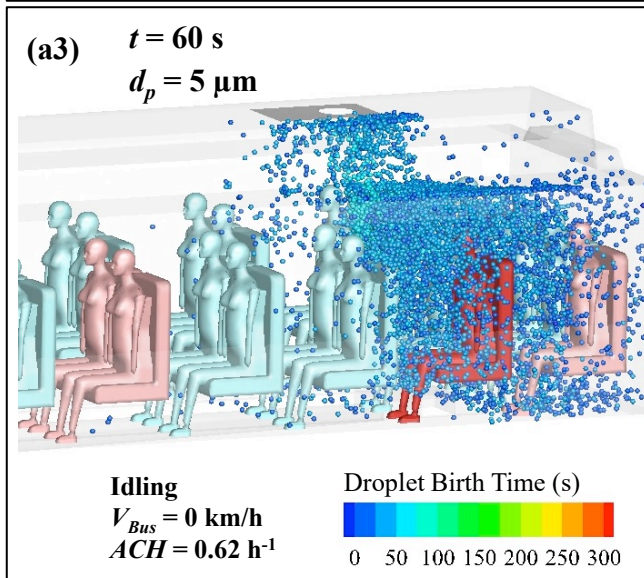
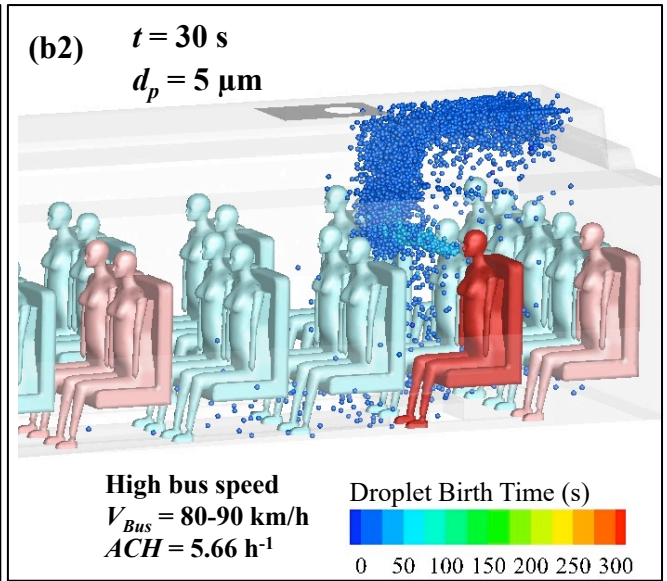
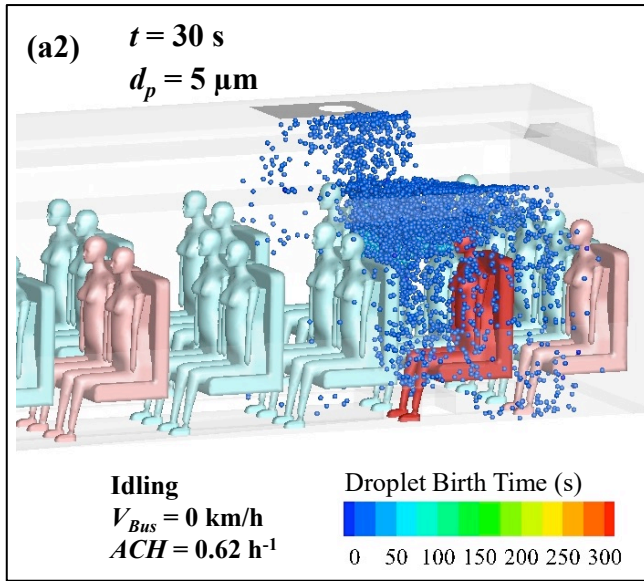
Fig. 6. Temporal variation of droplet evaporation ($d_p = 50 \mu\text{m}$, $100 \mu\text{m}$; RH = 35%, 95%).

3.2.2. Impacts of ventilation rates on droplet dispersion

We have investigated the impacts of ventilation rates on the dispersion of droplets in different initial diameters ($5\text{ }\mu\text{m}$, $50\text{ }\mu\text{m}$ and $100\text{ }\mu\text{m}$), and find that the dispersion mechanism is more affected by the gravity force and less influenced by the ventilation airflow for larger droplets. Thus, to better reveal the influence of ventilation rates, we only select $5\text{ }\mu\text{m}$ droplets to display their distribution under $RH = 35\%$ at $t=5\text{ s}$, 30 s , 60 s and 300 s with three ACH , as shown in Fig. 7.

As verified in Fig. 7a1-b1, after being exhaled by the index patient, droplets first move forward due to the initial exhalation flow, then rise up following the upward flow near the index patient, and spread with the main airflow routes (Fig. 7a2-b2). Due to the variation in ventilation rates, the spatial distribution of droplets also differs significantly (Fig. 7a3-a4). When $ACH = 5.66\text{ h}^{-1}$ with larger supply airflow blowing to the bus front, more droplets move forward and escape from the skylight outlet, leaving relatively fewer droplets in the bus rear (Fig. 7a4-b4). The results show that increasing the ventilation rate is beneficial to droplet dilution and excretion, and significantly reduces the droplet concentration near the index patient (i.e., seats 11D, 12C and 13D).





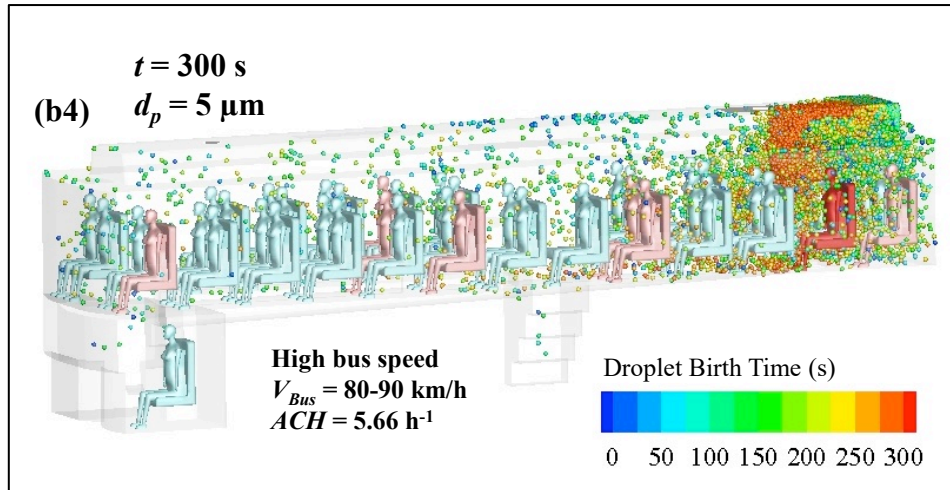


Fig. 7. Distribution of 5 μm droplets with $RH = 35\%$ at state of: (a) idling, (b) high bus speed.

3.2.3. Impacts of droplet initial diameters on droplet dispersion

Figure 8 indicates the evaporation and dispersion of droplets with different initial diameters when $RH = 35\%$ and $ACH = 2.27 \text{ h}^{-1}$. 5 μm droplets can evaporate rapidly into nuclei, so they are more significantly affected by the airflow field, and spread wider in the whole bus (Fig. 8a1-a3). Due to the combined action of airflow pattern and gravity force, 50 μm droplets mainly concentrate at the bus rear (Fig. 8b1-b3). With the dominance of gravity force, 100 μm droplets rapidly settle down from the exhalation jet after being exhaled from the index patient's mouth (Fig. 8c1-c3). Basically, the larger the initial diameter is, the quicker the droplets deposit, and hence the smaller range they propagate and the more they remain in the bus.

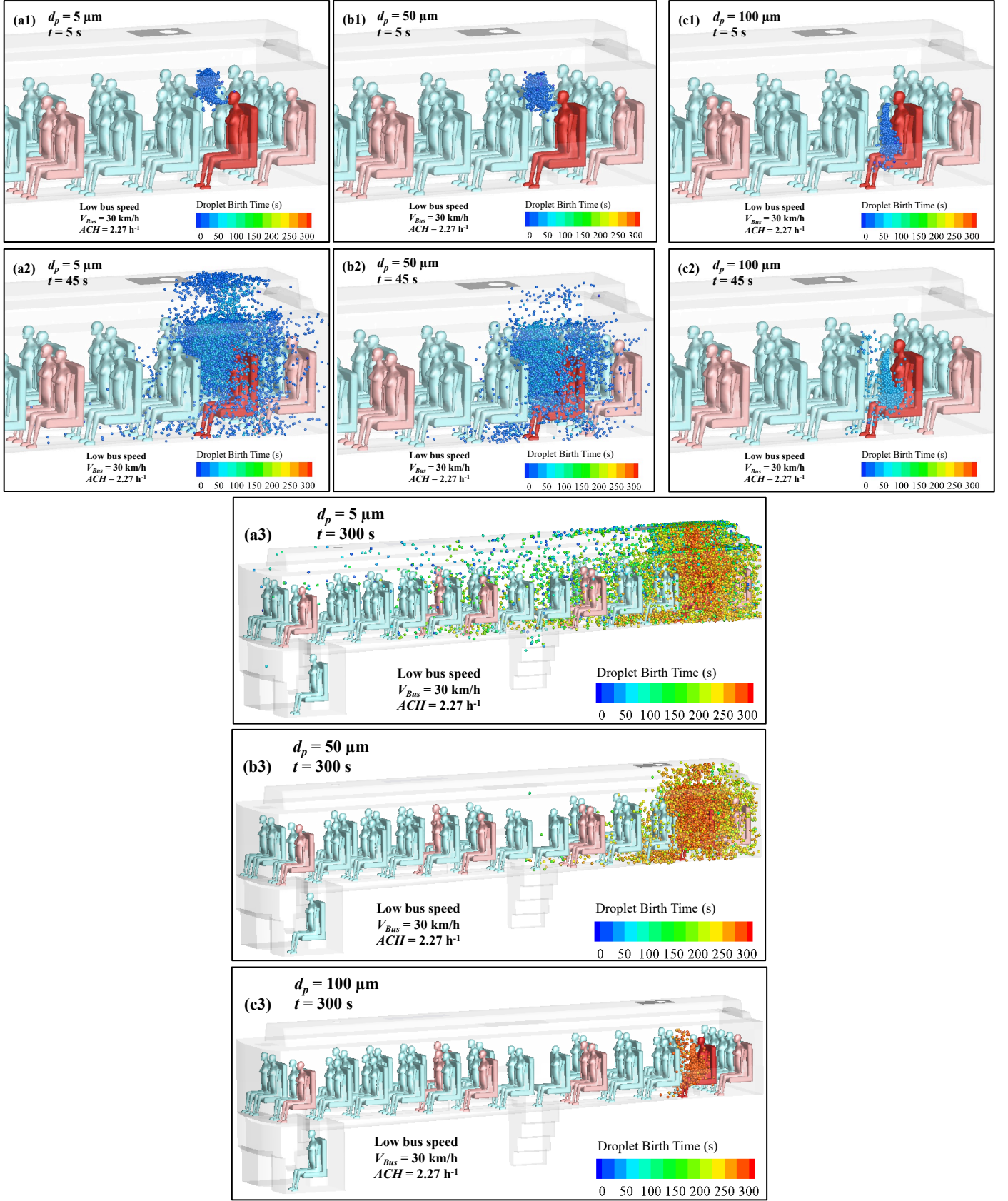


Fig. 8. Droplets distribution in low bus speed with $RH = 35\%$: (a) $d_p = 5 \mu\text{m}$, (b) $d_p = 50 \mu\text{m}$, (c) $d_p = 100 \mu\text{m}$.

3.3. Intake fraction and infection risk of each passenger

Fig. 9 depicts the 30-minute-exposure intake fraction (TIF) of each passenger which is defined as dividing the number of droplets a passenger inhaled by the total number of droplets released from the index patient (360,000). Fig. 10 depicts the 30-minute-exposure infection risk (TIR) of each passenger which is calculated by Wells-Riley equation (Eq. (9)). The X -axes represent the row of each passenger, where 12D is the location of the index patient and 8C is unoccupied. Passengers without data indicate that they did not inhale PLD released by the index patient.

For 5 μm droplets, the ventilation rates influence the TIF and the subsequent TIR , with a higher ventilation rate leading to more passengers at TIR . When $ACH = 0.62 \text{ h}^{-1}$, only few 5 μm droplets are inhaled by passengers in the bus front (Fig. 9a), leading to most front passengers at no droplet TIR (Fig. 10a). When ACH increases to 5.66 h^{-1} , even more front passengers are at TIR , and both TIF and TIR of passengers decrease with the distance between the passenger and index patient (Fig. 9c). Although 5 μm droplets disperse more widely with the increasing ventilation, the TIR is quite low ($<0.01\%$) for front passengers. Regardless of ventilation rate, more than 97% of 50 μm droplets deposit near the index patient due to gravity (Table. S1), so only the middle and rear passengers are at TIR with the highest infection risk for passenger 12C (3.13% under $ACH = 5.66 \text{ h}^{-1}$) (Fig. 10b). While for 100 μm droplets, over 99.5% of them deposit locally due to gravity (Table. S1), making nobody at TIF , so we don't display the infection risk.

For the tracer gas, a higher ventilation rate leading to lower TIR . The TIR is 11.10-15.29% under $ACH = 0.62 \text{ h}^{-1}$, and decreases to 1.27-3.09% when $ACH = 5.66 \text{ h}^{-1}$ (Fig.

10c). The *TIR* of tracer gas for each passenger is more uniform and distinctly higher under the same condition, compared with that of droplets.

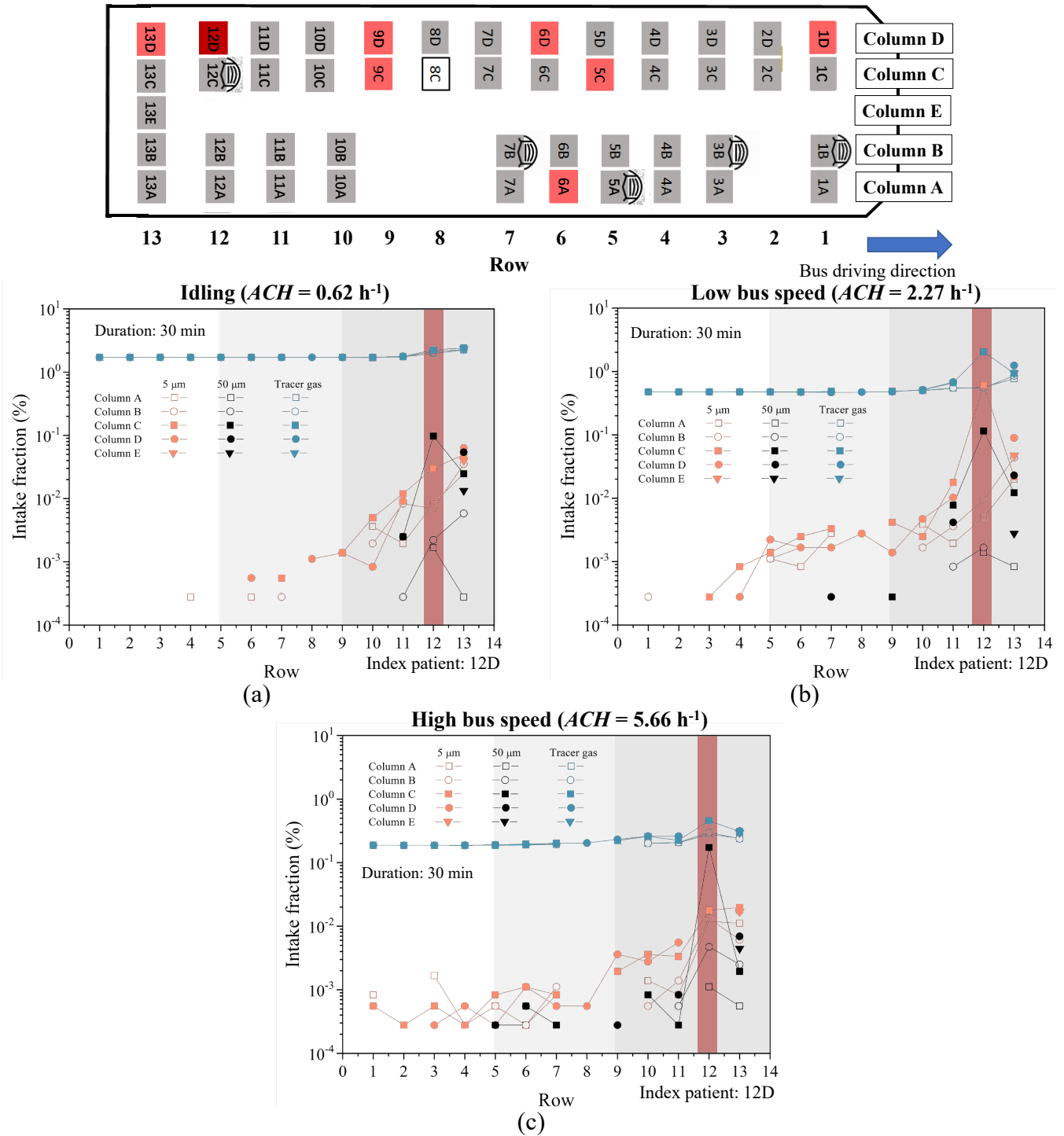


Fig. 9. Intake fraction of each passenger in a duration of 30 min under $RH = 35\%$ at state of: (a) idling, (b) low bus speed, (c) high bus speed.

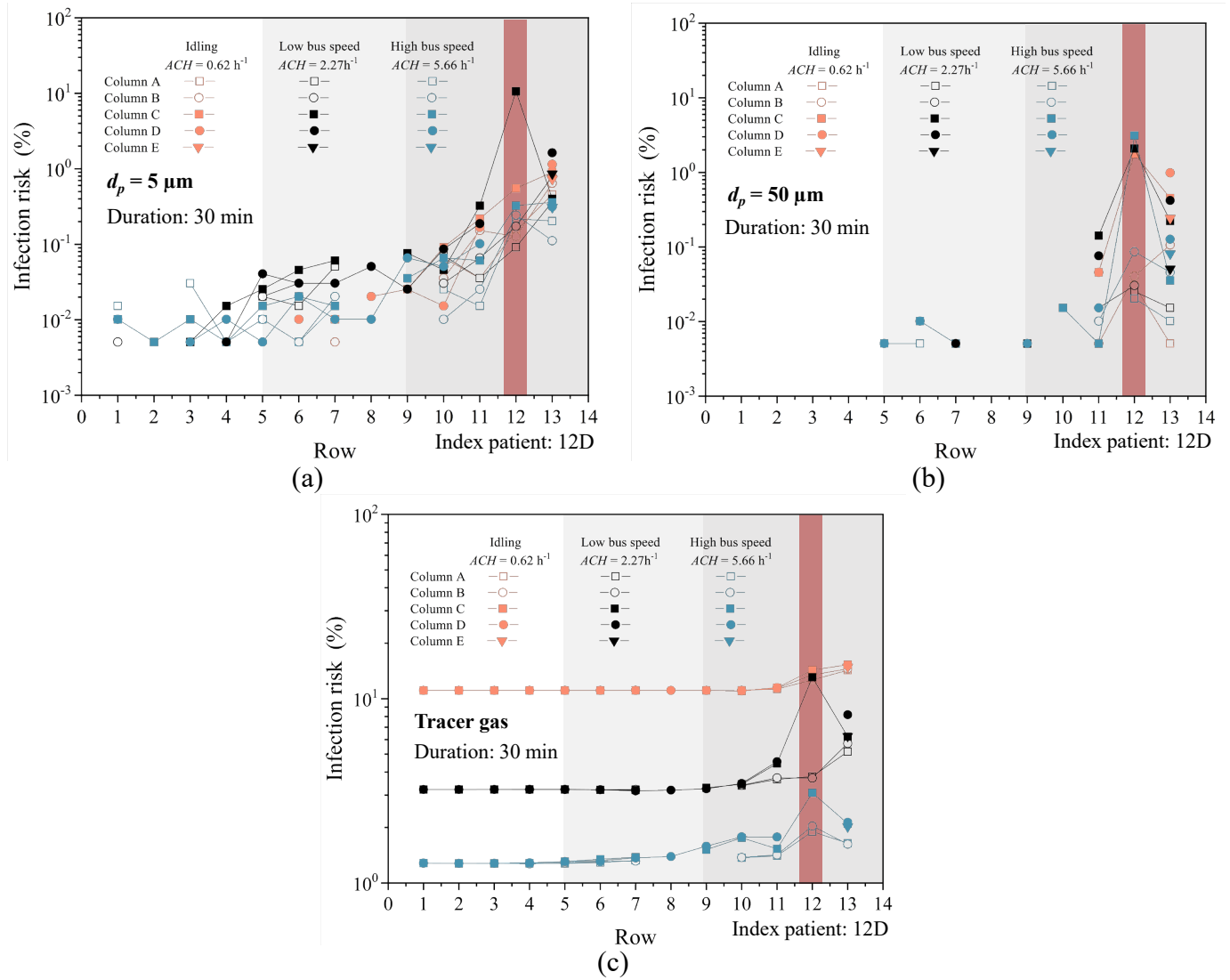


Fig. 10. Infection risk of each passenger under $RH = 35\%$: (a) $d_p = 5 \mu\text{m}$, (b) $d_p = 50 \mu\text{m}$, (c) tracer gas. (Note: infection risk within 30 min was calculated.)

416

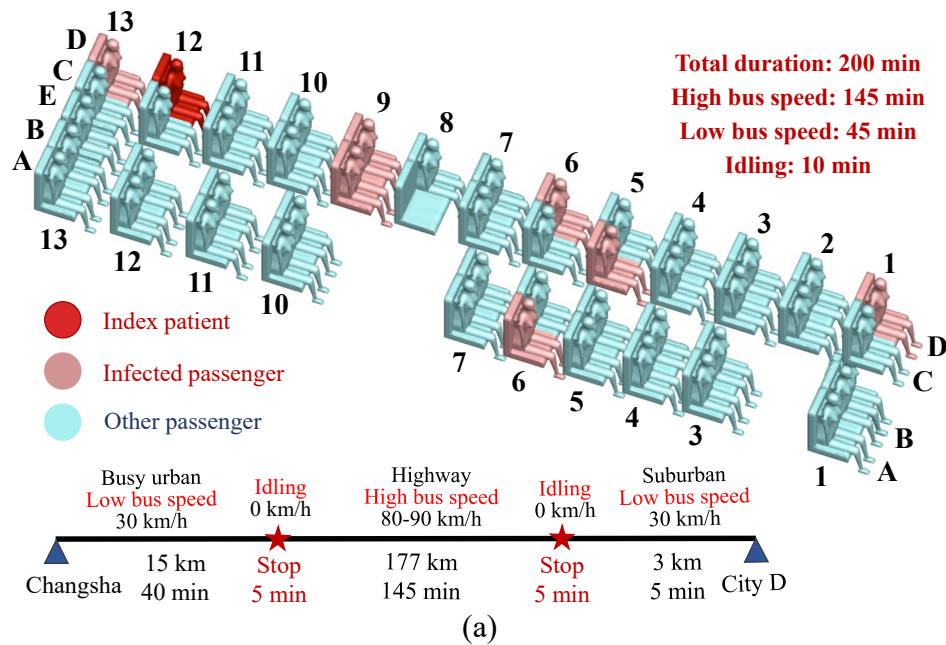
417 **3.4. Infection risk of each passenger in this epidemic outbreak**

418 The total duration of the bus journey is 200 min, and the passenger seating
 419 arrangement and driving route are depicted in Fig. 11a. We adopted the calculated 30-
 420 minute simulation results to infer the quanta of virus-laden droplets or tracer gas inhaled
 421 by each passenger throughout the whole journey. Fig. 11b-11d display the whole-

journal-exposure infection risk (*WIR*) of passengers (except the index patient at seat 12D). Note that the logarithmic coordinate system is employed in Fig. 11c-11d.

Under all conditions, the highest *WIR* of the tracer gas, 5 μm and 50 μm droplets occurs at seat 12C with 33.85%, 16.99% and 17.40%, and followed by seat 13D with 24.97%, 4.28% and 1.57%, respectively. It can be seen from Fig. 11 that the *WIR* of front-seat passengers significantly decreases with the increasing initial droplet diameter. However, passengers near the index patient (i.e., seats of 11D, 12C and 13D) are always at comparatively high *WIR*.

For the tracer gas (Fig. 11b), the *WIR* of front passengers is relatively even at ~14.00%. For 5 μm droplets (Fig. 11c), the *WIR* is quite discrepant for passengers at different locations, and is less than 0.15% for the front passengers (Rows 1-4), while up to 16.99% for the passenger at 12C. For 50 μm droplets (Fig. 11d), no front passengers are at *WIR*.



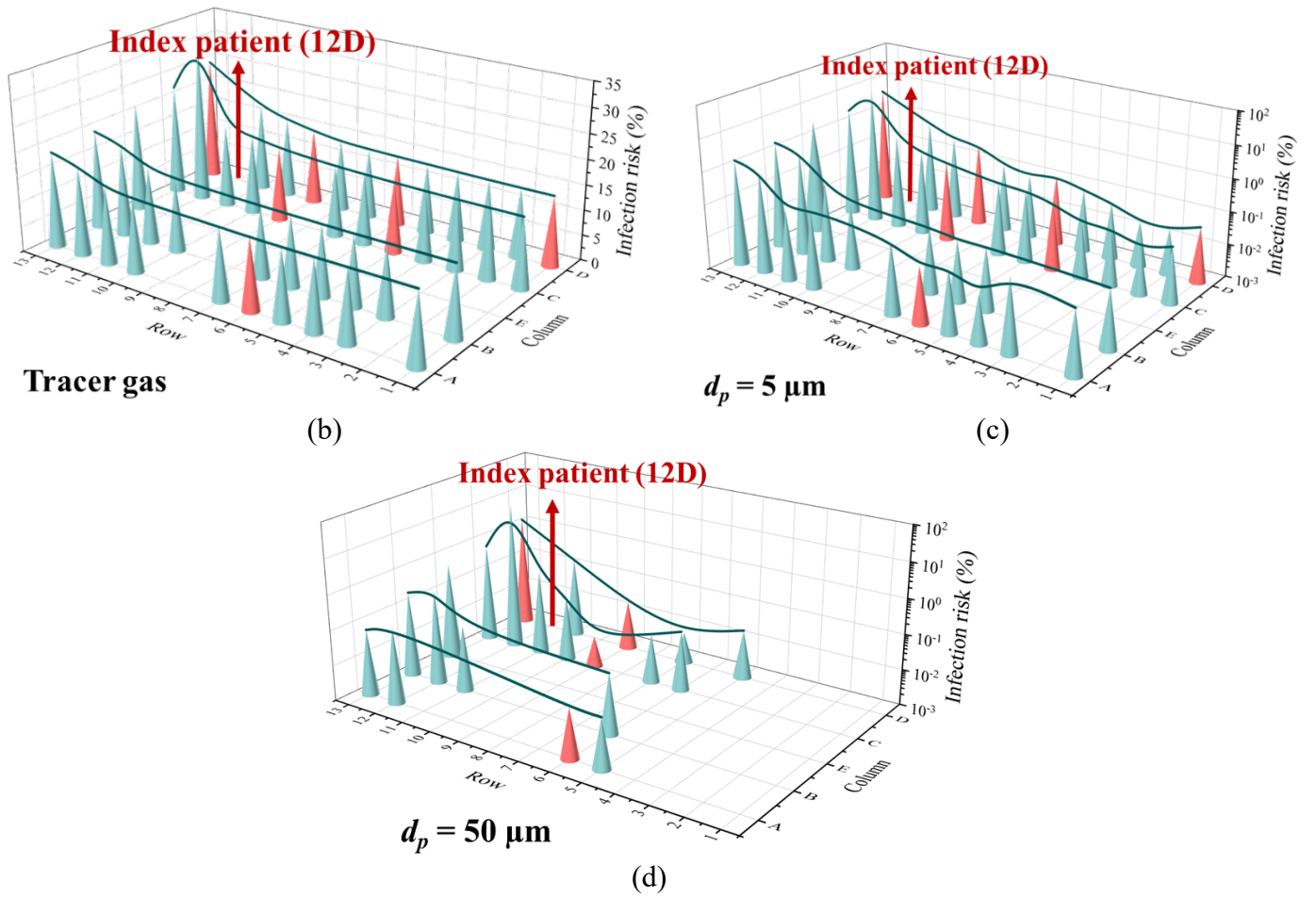


Fig. 11. (a) Seating arrangement, journeys, and distribution of index patient and infected passengers, (b) Tracer gas, (c) $d_p = 5 \mu\text{m}$, $RH = 35\%$, (d) $d_p = 50 \mu\text{m}$, $RH = 35\%$. (Note: infection risk is calculated based on this epidemic case.)

4. Discussion

4.1. The unique airflow field characteristics of the coach bus

The strength of this study lies in the investigation of the combined effect of the indoor main airflow and human thermal plumes on the airflow field in this unique in-coach environment. The coach bus only has small roof-opening skylight suppliers to open up for fresh air, but no windows are openable. Fig. 12 depicts the external surface wind pressure coefficient around the running bus. It shows that the pressure on the rear half is higher than the front half of the bus, which leads the air enter the bus from the

443 rear skylight and exit from the front, i.e., the indoor main airflow is moving from the
 444 rear to front. This unique rear-to-front airflow pattern makes the pathogen-laden
 445 expiratory droplets propagate the entire bus when the index patient is seated at the bus
 446 rear (12D) and hence results in large-scale transmission in this outbreak, as was also
 447 found in Mesgarpour et al. [58]. If the index patient was in the middle or front of the
 448 bus, the rear of the bus will be a low-risk area [59].

449

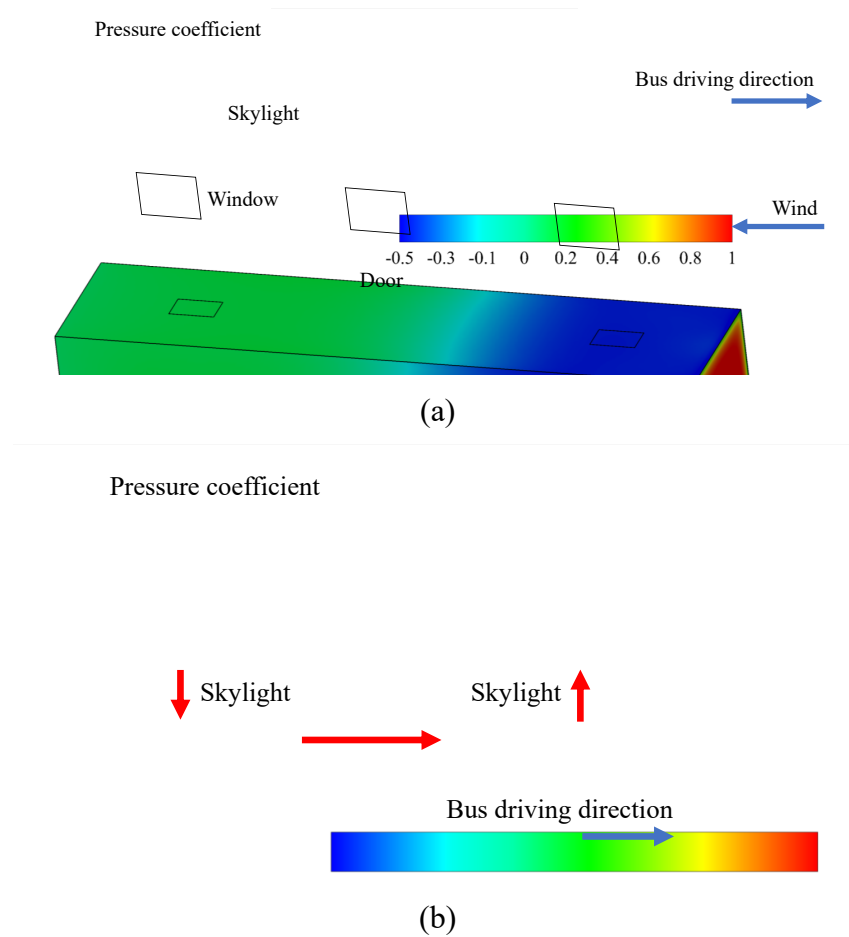


Fig. 12. (a) Pressure coefficient distribution on bus surfaces, (b) External pressure coefficient distribution of bus.

450

Besides the main airflow, the body thermal plumes cannot be neglected in the crowded coach bus, as they can cause an upward airflow near each human body and hence influence the droplet dispersion in the respiratory region—the last few inches for aerosol transmission to effectuate [17, 22, 60]. Meanwhile, Yang et al. [61] found that strong main airflow could destroy the thermal plumes. The body thermal plumes are obvious under the small background velocity field and can inhibit droplets from entering the respiratory area. This certain protective effect makes droplet infection risk stay low under $ACH = 0.62 \text{ h}^{-1}$. However, the body thermal plumes will be disturbed with the enhancement of the main airflow, leading to an increase of the droplet infection risk under $ACH = 2.27 \text{ h}^{-1}$.

4.2. Impacts of RH and initial droplet diameter on droplet dispersion

Redrow et al. [38] demonstrated that $10 \text{ }\mu\text{m}$ droplets could evaporate completely in 0.25 s at $RH = 20\%$ and 0.55 s at $RH = 80\%$, and RH influenced $0.4\text{-}10 \text{ }\mu\text{m}$ droplet transport in a simulated room where the mean air velocity was almost zero. Liu et al. [17] revealed that $100 \text{ }\mu\text{m}$ droplets took more than 100 s to evaporate at $RH = 95\%$ and $<2 \text{ s}$ at $RH = 35\%$, which made $100 \text{ }\mu\text{m}$ droplet dispersion totally distinct under different RH in an empty room. However, our study achieved a completely different finding: RH rarely influences the droplet ($5\text{-}100 \text{ }\mu\text{m}$) dispersion in the coach bus. The possible reason may lie in the complex indoor environment of the coach bus which is different from those in the above literature. In our study, we found that the interaction of the main airflow and body thermal plumes made the airflow much more complex, which significantly influenced the droplet/tracer gas dispersion. Moreover, Chen and Zhao [62]

and Xie et al. [33] indicated that regardless of the RH , small droplets evaporated completely quickly, and big droplets deposited downward immediately before fully evaporating due to gravity dominance. Therefore, there was a tiny difference of droplet ultimate fates and infection risk between different RH (Table. S1, Fig. S2), which agreed well with the study on coach bus conducted by Yang et al. [28].

The droplet diameter is the fundamental property that determines its transport characteristics. The transport behavior of a droplet depends on its interaction with the surrounding gas molecules, as well as the force acting on it [63]. When the droplet diameter increases, its dominant influencing mechanism changes into gravity force or drag force [64, 65]. Zhu et al. [66] indicated that the droplets of 30 μm or smaller were mostly influenced by indoor airflow, but those of 50 – 200 μm were significantly affected by the gravity force. Our study found that small droplets (i.e., tracer gas and 5 μm droplets) can follow the airflow and spread throughout the cabin, while large droplets (i.e., 50 μm and 100 μm) deposited near the index patient due to the dominant gravity force. Namely, small droplets can travel farther than large droplets, leading to a larger range of inhalation transmission.

When the droplet is small enough, the behavior of the droplets and the surrounding gas requires the kinetic theory of gases. Therefore, tracer gas was adopted as a surrogate for droplets and droplet nuclei smaller than 5 μm in general room environments, which had been verified by existing studies [36, 44, 67]. However, unlike general rooms, buses are longer and narrower in shape (11.4 m long and 2.5 m wide in our study) with more obstacles (i.e., human bodies and seats), which provides much more surface for droplets to deposit. Our study verifies that most droplets deposit on the route through the long-

and-narrow bus so that only a small fraction can spread to the bus front. Therefore, passengers in the bus front can expose to few droplets and lead to a quite low infection risk. However, tracer gas does not deposit and can disperse in the whole cabin, resulting in distinctly higher infection risk under the same condition. Hence, tracer gas cannot be utilized to mimic the dispersion processes of droplets which can be deposited on the surfaces. Meanwhile, Zhao et al.[68] indicated that the deposition of 0.7 μm particles was insignificant in an aircraft cabin. Lai and Nazaroff [69] reported that droplets in the range of 0.1-0.2 μm has the lowest deposition rate in indoor environments. Hence, we conclude that tracer gas can only be adopted to simulate the dispersion of fine droplets (e.g., 0.1-0.7 μm) with little deposition in coach buses.

4.3. Impacts of ventilation rates on droplet dispersion and infection risk

van Doremalen et al. [6] have found that the SARS-CoV-2 virus can remain infectious in aerosols for hours and up to days on surfaces, leading to probable transmission. Among the three main transmission routes, the aerosol inhalation route is predominant and can occur over a long distance when the ventilation is insufficient [64, 70]. Therefore, this study aims to investigate the mechanism of factors affecting the aerosol inhalation transmission and infection risk in a crowded coach bus.

Enhancing the indoor ventilation rates can promote dilution and removal of pathogen-laden expiratory droplets or droplet nuclei, and hence reduce the infection risk [10, 67, 71]. Our study also confirms that the infection risk is closely related to ventilation rates. When the ventilation rate is small, droplets can only disperse in the bus rear and middle. Larger ventilation airflow drives droplets to disperse more widely in the bus, but the infection risk is relatively low in the bus front (lower than 0.1% when $ACH = 5.66 \text{ h}^{-1}$ for 5 μm droplets). While for tracer gas, the inhalation infection risk can

be reduced by an order of magnitude as ACH increases from 0.62 h^{-1} to 5.66 h^{-1} . Thus, for the large range of initial diameters of respiratory droplets, the infection risk decreases with the increasing ventilation rates.

The World Health Organization [72] has indicated that the long-range aerosol inhalation transmission of COVID-19 is opportunistic in specific settings, particularly in crowded and inadequately ventilated indoor environments. Li et al. [10] revealed the long-range aerosol inhalation transmission in an insufficient ventilation restaurant and the longest transmission distance is 4.6 m. In this outbreak, the coach bus was supplied at a time-average ventilation rate of 1.72 L/s per person [11] far lower than the ASHRAE Standard (2019) [73], leading to long-range aerosol inhalation transmission with a longest distance of 9.46 m between the index patient and the infected passenger (1D). Both simulation results and actual outbreak confirm the important role of ventilation on aerosol inhalation transmission and infection risk.

4.4. Infection risk in realistic bus outbreak

Another merit of this study lies in that we utilized the real outbreak data to back-calculate the infection risk of each passenger according to the bus speeds and the corresponding exposure time in this COVID-19 outbreak inside the coach bus. Based on the numerical calculation results, we explained the following three characteristics for the spatial distribution of infected passengers in this realistic epidemic: (1) more infected passengers in the middle and rear of the cabin (six in Row 5-13) than in the front (only one in Row 1-4); (2) more infected passengers on the index patient side (six in Column C-D) than on the opposite side (only one in Column A-B).

The trajectory of droplets is determined by the airflow pattern, gravity force, and the process of evaporation in terms of their diameter. $50\text{ }\mu\text{m}$ droplets can transmit a

short distance and then gradually deposit due to the gravity force, so only part of them can be inhaled by passengers in the rear and middle, which leads to short-range aerosol inhalation transmission [7]. Meanwhile, smaller droplets ($\leq 5 \mu\text{m}$) can continue spreading to the bus front, leading to both short and long-range aerosol inhalation transmissions. Thus, the infection risk is higher near the index patient and decreases with distance, namely, more passengers in the middle and rear were infected than those in the front.

Higher infection risks for both tracer gas and droplets are found on the index patient's side (Column C and D). The reason may lie in the cold "gravity current" [74] falling to the cabin floor and spreading throughout the entire cabin, established by the skylight inlet above the aisle near the index patient. The cold "gravity current" then rose with the body thermal plume of each passenger and finally exhausted at the bus front skylight outlet. The blockage of the floor-level "gravity current" by the toilet in the lower deck of the cabin made contaminated air spread slightly more to the index patient side than to the opposite side, which brought about a higher infection risk on the index patient side.

It can be found that there are some deviations between the realistic location of the infected person and the calculated location of passengers at high infection risk. The reason may lie in the following aspects. Firstly, we have calculated the infection risk of aerosol inhalation transmission for droplets with some representative diameters (tracer gas, $5 \mu\text{m}$, $50 \mu\text{m}$, and $100 \mu\text{m}$). However, the PLD is in the large range of diameters ($0.1 - 100 \mu\text{m}$) which may highly affect the infection risk. Thus, it needs further study on the infection risk distribution of droplets in large range of diameters. Secondly, there are many other factors that may affect passengers being infected, such as other

transmission routes (e.g., direct-contact/indirect-contact transmission [75]), immunity of passengers, the activity status of passengers in the bus (wear a mask or not, speak or not, etc). However, our study quantified the impact of natural ventilation on the infection risk of tracer gas and droplets and provided a basis for the prevention and control of respiratory infectious diseases in coach buses.

4.5. Limitations and future research

There are several limitations of the present research that should be acknowledged. Different respiratory activities, including breathing, speaking, coughing, sneezing, etc, can affect the generation and dispersion of droplets [26, 58, 76]. We only considered the index patient's breathing activity, because the epidemiological survey suggests that the index patient did not cough or talk to anyone during the whole trip. In the future, we will further consider more respiratory activities and more influencing parameters (e.g., natural ventilation modes by opening windows, source location, ambient temperature, different total heat flux for occupant etc). Meanwhile, it deserves further investigation on how the droplet final fates change with various ventilation rates and initial diameters under the combined effect of ambient airflow, gravity and body thermal plumes. Due to the positive pressure at the bus rear and the negative pressure at the bus front, opening the windows at the bus rear is beneficial to increase the ventilation rates, but the specific method needs further evaluation. Additionally, during the epidemic of infectious disease, public vehicles are required to be less than half occupancy in order to reduce the infection risk, which has not only caused great economic losses to the transportation operation companies, but also caused inconvenience to people's travel. Therefore, it is worth further investigating the infection risk under different seat

arrangements to give a more specific suggestion for arranging the occupancy in different coach buses.

5. Conclusions

Based on experiments of one-infecting-seven COVID-19 outbreak with an index patient at bus rear, this paper performed CFD simulations to explore the PLD dispersion and infection risk in a crowded coach bus, which is important but still scarce. The integrated effects of initial droplet diameters, natural air change rates per hour and relative humidity are considered.

Some meaningful conclusions can be stated:

- (1) In this bus epidemic, inadequate ventilation, crowded passengers and long exposure time (200 min) are the main reasons for the large number of infected passengers (i.e., seven) with a longest infected distance of 9.46 m.
- (2) The pressure difference between the bus rear and front makes the air enter the bus from the rear ceiling-level skylight (inlet), and leave through the bus front ceiling-level skylight (outlet), carrying droplets/tracer gas disperse from the rear to the front. Higher bus speed leads to more ventilation rates.
- (3) Tracer gas can only be adopted to simulate fine droplet (e.g., 0.1-0.7 μm) dispersion in coach buses. The gaseous inhalation transmission can occur in the entire cabin, and its infection risk is greatly reduced with the increasing ventilation rates. When ACH increases from 0.62 h^{-1} to 2.27 h^{-1} and 5.66 h^{-1} , TIF of tracer gas for each passenger decreases from 11.10-15.29% to 3.20-13.08% and 1.27-3.09%.

(4) Over 99.5%/97.0% of large droplets (i.e., 100 μ m/50 μ m) deposit locally due to gravity. Thus, the *TIF* of 100 μ m/50 μ m droplets is almost independent of *ACH*, with a peak *TIF* (~3.1%) near the index patient. Because gravity is less significant for 5 μ m droplets which can spread more widely with the ventilation airflow from bus rear to front and disperse even further with the increasing ventilation.

(5) Unlike ordinary rooms, most droplets will deposit on objects when spread in the long-and-narrow bus, but tracer gas will not deposit. therefore, the infection risk of tracer gas is obviously higher than that of 5-100 μ m droplets.

(6) Relative humidity (*RH*=35% and 95%) affects the droplet evaporation process, but insignificantly influences the dispersion and infection risk.

For coach buses and other indoor environments, fresh air should be sufficiently supplied for the occupants. When the occupancy rate of vehicles is high, it is recommended to open windows or ceiling-level skylight at the vehicle rear to attain more fresh air into the bus and better natural ventilation. Even for cold winter or hot summer, we should find a balance between energy consumption and human health, i.e., to ensure thermal comfort by air conditioners or heating devices meanwhile provide sufficient external fresh air by opening ceiling-level skylight or small-area windows to reduce the infection risk in the vehicle.

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