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Residence Time Distribution Analysis of Patient-Emitted Air in a Mock Hospital Isolation Room: A CFD Study

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Abstract

Air changes per hour (ACH) is the primary metric specifying ventilation in hospital isolation rooms, yet presumes the room behaves as a perfectly mixed reactor. However, short-circuiting pathways and recirculation (dead) zones make the removal of patient-discharged air strongly dependent on airflow topology. This study applies residence time distribution (RTD) analysis to quantify that dependence. Computational fluid dynamics (CFD) simulations of a mock hospital isolation room were performed at 6 and 12 ACH in two exhaust configurations: a normal design (NMD), with the exhaust displaced from the patient, and a short-circuiting design (SCD), preserving a direct patient-to-exhaust pathway that acts as source capture. A passive tracer pulse released at the patient mouth was tracked to the exhaust, giving the breakthrough time, the 10–90% removal spread, and the mean residence time, each normalized by the theoretical mean residence time. At 6 ACH both configurations stayed close to the perfectly mixed prediction. At 12 ACH they diverged sharply: SCD reduced normalized breakthrough time from 0.311 to 0.0283, narrowed normalized spread from 2.29 to 1.64, shortened mean residence time from 6.23 to 2.64 min, and cut long-term healthcare worker exposure from 1.27 to 0.42 times the perfectly mixed value. Doubling ACH reduced that exposure by 75% under SCD but only 39% under NMD, and shortened early exposure only where the direct pathway was preserved. ACH specifies how much air is supplied, not where it goes; RTD metrics capture that difference and are obtainable from CFD or physical tracer testing.

Keywords: Hospital isolation room; Ventilation performance metrics; Residence time distribution; Air changes per hour; Computational fluid dynamics

1 Introduction

Computational fluid dynamics (CFD) in occupational safety enables researchers to identify factors that influence the dispersion of airborne substances hazardous to health or comfort and to evaluate potential exposure. In clinical environments, this can include exhaled air carrying infectious aerosols, whose spread poses risks to patients and healthcare staff. Accordingly, CFD investigations of airborne transport in healthcare settings [1] have often been motivated by outbreaks of respiratory viruses, such as the 2002–2004 SARS outbreak, the 2009 H1N1 influenza pandemic [2, 3], and, more recently, the COVID-19 pandemic [4]. While CFD can be computationally demanding, the increasing availability of computing power, combined with the relatively compact size of hospital isolation rooms compared with entire wards or buildings, makes it feasible to capture the transport and mixing of exhaled air under the influence of thermal and fluid-dynamic processes [5-7]. CFD provides predictions of three-dimensional airflow and tracer transport over time, yielding insight into flow features associated with particular ventilation layouts and room configurations. By examining how these structures govern the transport and dilution of patient-discharged air, the potential exposure of occupants can be evaluated and used to inform ventilation design.

This work uses CFD simulations to study airflow and patient-discharged air in a mock isolation room modeled after the configuration investigated by Thatiparti et al. [8, 9]. The room, patient, and healthcare worker (HCW) geometries are based on those studies. The analysis focuses on the short-circuiting or channeling pathway between the patient's mouth and the exhaust air vent, as sketched in Figure 1. When this pathway is preserved, air discharged from the patient can travel relatively directly to the exhaust rather than first dispersing throughout the room, potentially reducing its transport toward the HCW.

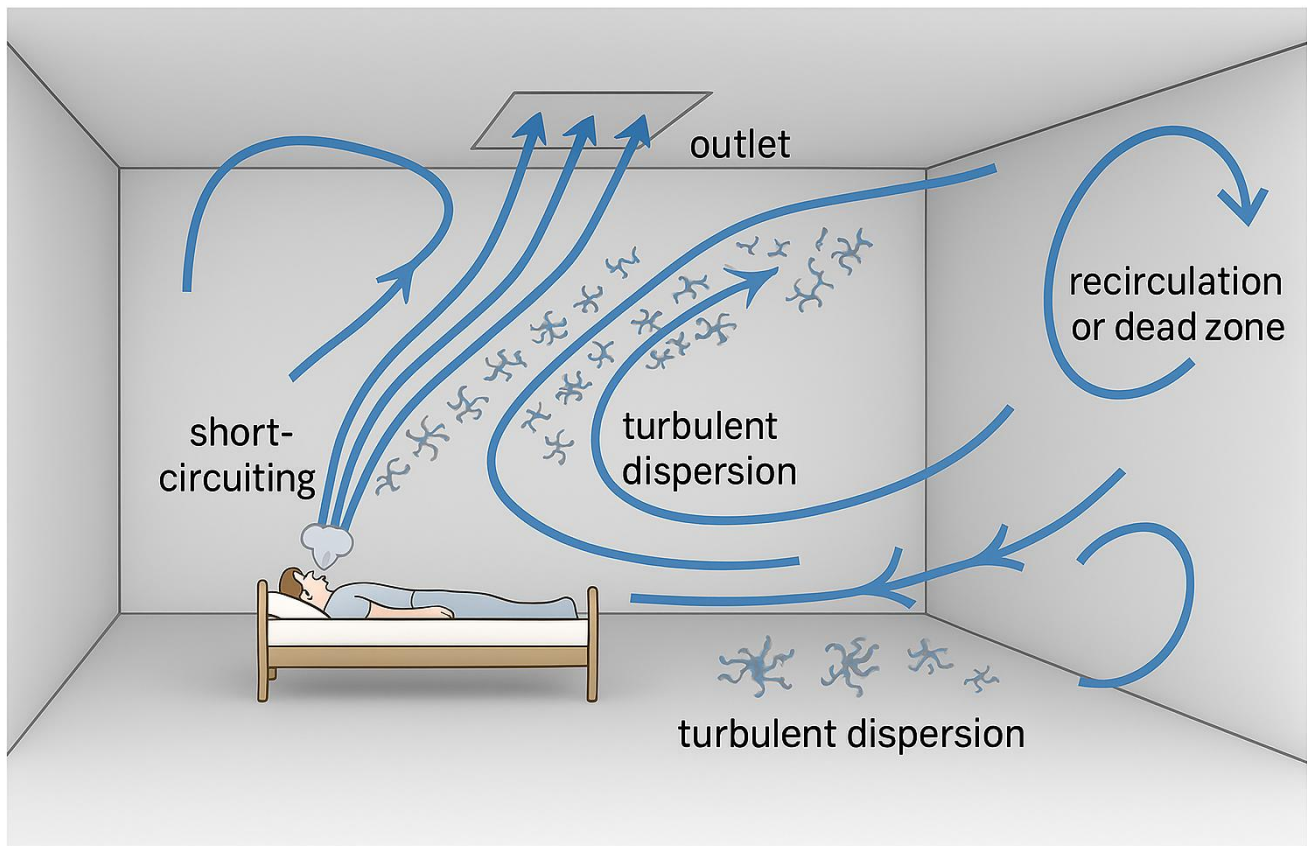


Fig. 1 Sketch of short-circuiting between the patient and the air exhaust (or outlet), recirculation or dead zones and turbulent diffusion (dispersion) in a hospital room.

Short-circuiting is often viewed as undesirable in conventional ventilation applications because supply air may pass directly to an exhaust without effectively ventilating the occupied space. The pathway considered here is different because it begins near the contaminant source and can act as a form of source capture. Previous CFD studies have shown that such patient-to-exhaust pathways can reduce HCW exposure to exhaled contaminants [5, 6, 10]. However, rapid transport through a short-circuiting pathway can coexist with recirculation regions or dead zones, such as those illustrated near the room corners in Figure 1. While a fluid parcel within a direct pathway can travel rapidly from the patient to the exhaust, a parcel entering a recirculation region may remain in the room until it is transferred back to

the primary flow by turbulent dispersion. Thus, the overall transport and removal of patient-discharged air reflect the combined influences of organized advection, recirculation, and turbulent dispersion.

A widely used model for estimating the removal of a pulse of exhaled air from a ventilated room is based on the classical perfectly mixed reactor assumption (PMA) [11]. This model assumes that the discharged air is instantaneously and uniformly distributed throughout the room after release. For a room of volume V ventilated at a volumetric flow rate Q , the theoretical mean residence time is $\tau_{theor} = V/Q$. Under perfectly mixed conditions, the room-average concentration decays according to $C(t) = C_0 \exp(-t/\tau_{theor})$, where C_0 is the initial concentration. The corresponding decay rate is related to the room air changes per hour (ACH).

Actual mixing conditions in hospital rooms often deviate from this ideal because of short-circuiting pathways and recirculation zones, as illustrated schematically in Figure 1. These flow structures can locally retain patient-discharged air or enable its rapid removal, leading to substantial spatial and temporal variations in concentration and HCW exposure. For example, Memarzadeh and Xu [5] demonstrated through CFD that a strong patient-to-exhaust pathway can substantially reduce HCW exposure, an effect that cannot be represented by a perfectly mixed model.

The limitations of the perfectly mixed assumption have been recognized in recent research, which has shown that well-mixed conditions alone are inadequate for assessing localized exposure in environments characterized by complex airflow and temperature stratification [6, 12]. Nevertheless, ACH remains widely used in HVAC engineering practice and is incorporated into standards such as ASHRAE 62.1 and ASHRAE 170 [13, 14]. During the COVID-19 pandemic, for example, the U.S. Centers for Disease Control and Prevention recommended five or more ACH to mitigate airborne transmission of SARS-CoV-2 [15]. More broadly, ACH has long served as an important design criterion for healthcare ventilation systems [16, 17]. ACH is a bulk measure of ventilation rate; however, using it

to predict a uniform concentration-decay rate or contaminant-removal time implicitly assumes that the room behaves as a perfectly mixed system.

The finding of Memarzadeh and Xu [5] that a direct patient-to-exhaust pathway can reduce HCW exposure was based on CFD simulations over a five-minute exposure window. Many HCW activities extend beyond this duration. For example, nurses spend, on average, more than one hour per shift inside patient rooms [18]. Understanding and minimizing cumulative exposure to patient-discharged air over longer periods is therefore also important for ventilation design, particularly in environments occupied by patients with respiratory infections [19, 20].

Residence time distribution (RTD) analysis has long been used in chemical-reactor and drinking-water-disinfection studies to characterize nonideal transport, short-circuiting, and retention [21-24]. This study makes use of the RTD, for the first time to the authors' knowledge, as a means of formally evaluating the transport and removal of patient-discharged air in a hospital isolation room. RTD analysis can be performed using CFD simulations or physical tracer experiments, allowing it to be applied virtually during design or empirically during commissioning and validation. In the present application, RTD and cumulative-RTD curves are used to quantify early exhaust breakthrough, the breadth of the tracer-removal response, and overall residence time. These measures characterize the observable consequences of the combined transport processes; they do not separately partition advection, recirculation, and turbulent dispersion.

The RTD analysis also provides a room-scale framework for interpreting HCW exposure. HCW exposure and cumulative RTD are both based on time integrals of tracer concentration, but they are not equivalent quantities. HCW exposure is based on concentration sampled locally near the HCW, whereas cumulative RTD is based on tracer leaving through the exhaust. It is therefore useful to examine how the exposure behavior observed in the simulations varies in relation to the cumulative-RTD characteristics,

while recognizing that local exposure also depends on the spatial arrangement of the patient, HCW, air supply, and exhaust.

The goals of this study are to:

1. Apply RTD and cumulative-RTD analysis to CFD simulations of a mock hospital isolation room to characterize the combined influence of short-circuiting, recirculation or dead zones, and turbulent dispersion on the transport and removal of patient-discharged air.
2. Evaluate how ventilation performance and HCW exposure vary with ACH and exhaust placement in normal and short-circuiting room designs, and compare the CFD results with predictions based on the perfectly mixed assumption or PMA.
3. Examine how short- and long-term HCW exposure vary in relation to CFD-derived RTD metrics and determine whether these metrics provide a useful complementary framework for assessing hospital-room ventilation beyond ACH alone.

2 Methods

2.1 RTD analysis

In the classical approach to RTD analysis [24], a passive tracer (i.e. a scalar) pulse is released at the inlet of a stirred tank reactor and the tracer concentration is measured at the outlet over time yielding the so-called RTD curve (e.g. see sketch in Figure 2a). The RTD curve can be used to calculate residence time characteristics of the tracer in the reactor such as mean residence time (MRT) and can be used to quantify the importance of short-circuiting and dead zone flow features on the transport of tracer throughout the reactor. In the case of the isolation room studied here, once a steady state airflow solution inside the room has been obtained, RTD analysis is performed by releasing a pulse of air from above the mouth of the patient (Figure 1), tracked as a passive tracer. The steady state flow is used to transport the

tracer throughout the isolation room while the time series of the tracer concentration, $C(t)$, is recorded at the exhaust air vent or outlet to obtain the RTD curve.

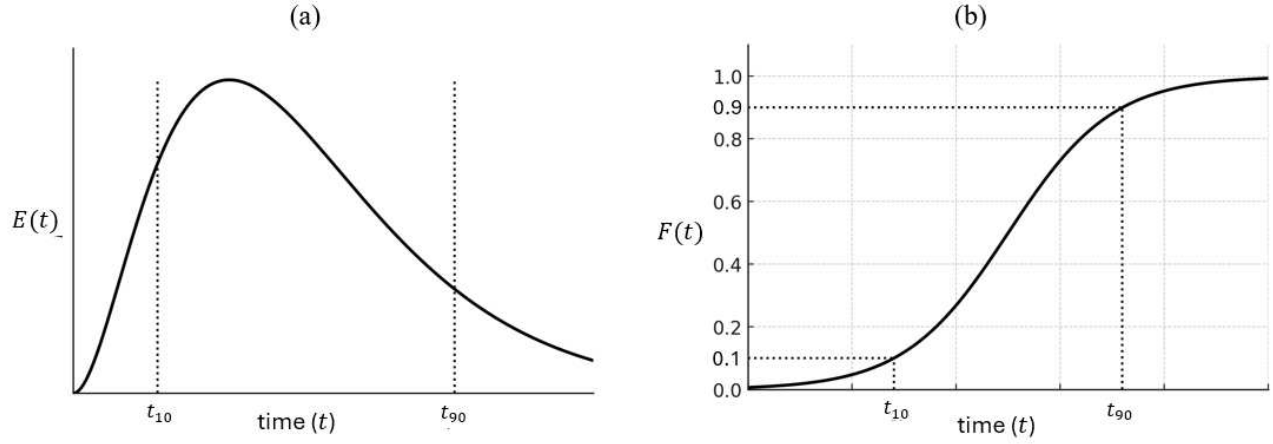


Fig. 2 Sketch of (a) RTD function $E(t)$ and (b) cumulative RTD function, $F(t)$, for an arbitrary system.

The RTD curve is defined as the normalized $C(t)$:

$$E(t) = \frac{C(t)}{\int_0^{\infty} C(t) dt} \quad (1)$$

As sketched in Figure 2a, the RTD curve rises from zero, reaches a peak, and then decays as tracer parcels are gradually exhausted. The initial rise of $E(t)$ is closely linked to the early exit of tracer along short-circuiting pathways. Accordingly, the breakthrough time t_{10} , as the time when 10% of the tracer has exited, occurs on the rising limb of $E(t)$ and quantifies early breakthrough due to short-circuiting.

The tail of $E(t)$ reflects tracer that lingers in recirculation (dead-zone) regions and is released gradually by turbulent dispersion (diffusion). Quantitatively, tail prominence can be characterized by the spread window $(t_{90} - t_{10})$ and the Morrill index t_{90}/t_{10} where t_{90} is the time when 90% of the tracer has exited. Larger values of the spread index indicate broader, diffusion-dominated removal. The Morrill index compactly captures the combination of early breakthrough and late-time persistence: all else

equal, reducing t_{10} increases t_{90}/t_{10} . Thus, a larger Morrill index indicates stronger short-circuiting (smaller t_{10}) and/or a more persistent tail (larger t_{90}); which factor dominates can be diagnosed by examining $(t_{90} - t_{10})$ alongside t_{10} . For comparisons across ACH, we also report the normalized spread $W \equiv (t_{90} - t_{10})/\tau_{theor}$.

The first moment of the RTD curve provides the mean residence time

$$\tau = \int_0^{\infty} t E(t) dt \quad (2)$$

[24], capturing the influence of both short-circuiting and recirculation on mean retention. As noted in the Introduction, the theoretical MRT is the ratio of the volume of the room to the volumetric air flow rate entering and exiting the room:

$$\tau_{theor} = V/Q \quad (3)$$

The theoretical MRT corresponds to perfectly mixed conditions, however, as highlighted in the Introduction, mixing conditions within an isolation room deviate from perfect due to the presence of short-circuiting and dead zones. The strengths or influence of these flow features determine how much greater or lower the actual MRT is compared to theoretical MRT. For example, strong short-circuiting implies that the tracer will exit the room rapidly, leading to a lower actual MRT than the theoretical MRT. Weaker short circuiting and stronger dead zones imply that the dead zones will be able to retain elevated tracer concentrations within the room for greater periods of time. Overall, the expression in Equation (2) provides the actual MRT under the influence of the true mixing conditions that determine the RTD. As such, the CFD-computed RTD can be used to obtain the actual simulated MRT, to be referred to as τ_{CFD} in the discussions below. Furthermore, it is then useful to define an apparent dead volume (inactive volume) fraction,

$$\phi_d = 1 - \frac{\tau_{CFD}}{\tau_{theor}} \quad (4)$$

which represents the fraction of the nominal volume not effectively contributing to tracer transport to the outlet. A positive ϕ_d indicates bypass or short-circuiting that reduces the effective residence time, while values closer to zero indicate behavior approaching ideal mixing.

The RTD function, $E(t)$, can provide further insight into the nature of the flow in the system through the cumulative RTD defined as

$$F(t) = \int_0^t E(t') dt' \quad (5)$$

[24]. Firstly, the cumulative RTD can be used to calculate the time it takes a certain percentage of the tracer to exit the room, as depicted in Figure 2b. From the sketch of Figure 2b and the definition in Equation (5), it can be seen that t_{10} (defined earlier as the time when 10% of the tracer exits the room) corresponds to the time at which $F = 0.1$. Similarly, t_{90} corresponds to the time at which $F = 0.9$.

As noted by Wilson and Venayagamoorthy [23], the shape of the cumulative RTD curve is indicative of the type of flow in the system. For example, a steeper gradient in the $F(t)$ curve corresponds to flow conditions closer to plug flow, which is dominated by advective transport relative to diffusion. A flow characterized by strong short-circuiting would give rise to a steeper gradient in $F(t)$, due to the strong advection (flow transport) characterizing the short-circuit. A flatter gradient in $F(t)$ is indicative of a flow dominated by diffusion relative to advective transport. The latter would correspond to a flow tending towards a perfectly mixed system.

The spread window ($t_{90} - t_{10}$) can be used as an index to help characterize the shape of $F(t)$, and thus the flow condition. A larger value of ($t_{90} - t_{10}$) would correspond to a flatter gradient in $F(t)$ and thus a flow dominated by diffusion, while a smaller value would correspond to a steeper gradient and thus a flow dominated by advection or short-circuiting.

Together, the indices ϕ_d , t_{10} , $(t_{90} - t_{10})$, and t_{90}/t_{10} associated with the RTD and cumulative RTD curves provide complementary measures of recirculation, short-circuiting, diffusion and advection. These metrics form the basis for the comparative analysis of ventilation performance of the hospital isolation room presented in Section 3.

2.2 Reynolds-averaged flow equations

The core of CFD consists of solving the fluid flow equations numerically. In this study, the equations of fluid motion consist of the compressible Reynolds-averaged continuity and the Reynolds-averaged Navier-Stokes (RANS) equations. These two equations are given using indicial notation in (6) and (7), respectively:

$$\frac{\partial \rho}{\partial t} + \frac{\partial}{\partial x_i}(\rho u_i) = 0 \quad (6)$$

$$\frac{\partial}{\partial t}(\rho u_i) + \frac{\partial}{\partial x_j}(\rho u_i u_j) = -\frac{\partial p}{\partial x_i} - \frac{\partial \rho \langle u'_i u'_j \rangle}{\partial x_j} + \frac{\partial}{\partial x_j} \left(\mu \left(\frac{\partial u_i}{\partial x_j} + \frac{\partial u_j}{\partial x_i} - \frac{2}{3} \delta_{ij} \frac{\partial u_l}{\partial x_l} \right) \right) + \rho g_i \quad (7)$$

where ρ is the fluid (air) mean density; p is mean pressure; μ is dynamic molecular viscosity; u_i is the mean velocity in the i -th direction; x_i is the coordinate in the i -th direction (the x , y , or z direction) in Cartesian components; g_i is the gravity vector taken to point in the negative z direction; t is time.

The Reynolds (or turbulent) stress in (7), defined as $-\rho \langle u'_i u'_j \rangle$, is induced by the turbulent nature of the flow field, and is defined in terms of the ensemble average (denoted by the brackets) of the product of fluctuating (turbulent) velocity components u'_i . Note that as per the classical Reynolds decomposition, the total velocity is the mean velocity, u_i , plus the fluctuating velocity u'_i . Following the well-known RANS simulation methodology employed here, the mean velocity u_i is computed or resolved via (6) and (7) while the Reynolds stress is modeled via the eddy (or turbulent) viscosity assumption as

$$-\rho\langle u'_i u'_j \rangle = \mu_t \left(\frac{\partial u_i}{\partial x_j} + \frac{\partial u_j}{\partial x_i} - \frac{2}{3} \delta_{ij} \frac{\partial u_l}{\partial x_l} \right) - \frac{2}{3} \rho k \delta_{ij} \quad (8)$$

In order to calculate the eddy viscosity, μ_t , and the turbulent kinetic energy, k , appearing in (8), the four-equation transition SST turbulence model is employed [25]. This model was chosen for its ability to accurately capture low free stream turbulence environments as well as swirl flows which are important characteristics in ventilation flows, such as those considered here.

The flow mean temperature, T , is calculated via

$$\rho C_p \frac{\partial T}{\partial t} + \rho C_p \frac{\partial u_i T}{\partial x_i} = - \frac{\partial}{\partial x_i} \left(\kappa_e \frac{\partial T}{\partial x_i} \right) \quad (9)$$

Note that the mean density in (6)-(9) is obtained from the mean temperature through the ideal gas law.

In (9), the effective conductivity κ_e is the sum of molecular and turbulent conductivities, with the turbulent conductivity computed through the eddy viscosity obtained from the transition SST closure.

2.3 Tracer concentration transport equation

The tracer released above the patient's mouth for RTD analysis is tracked via the convection-diffusion equation

$$\frac{\partial \rho C}{\partial t} + \frac{\partial \rho u_i C}{\partial x_i} = \frac{\partial J_i}{\partial x_i} + \rho S \quad (10)$$

where C is the tracer concentration, J_i the tracer diffusion flux, and S the tracer source.

The diffusion flux is defined as

$$J_i = \left(\rho D_m + \frac{\mu_t}{S_{ct}} \right) \frac{\partial C}{\partial x_i} + \frac{D_T}{T} \frac{\partial T}{\partial x_i} \quad (11)$$

where D_m and D_T are the tracer diffusion coefficient and the thermal diffusion coefficient, respectively.

The turbulent Schmidt number is taken as $S_{ct} = 0.7$.

Although the intent here is to perform a classical RTD analysis which is based on a passive tracer release (as described above), it is worth mentioning that Equation (10) has been shown to be a good

model for the transport of aerosol droplet nuclei corresponding to particles less than 20 μm in diameter [26]. Thus, the tracer could be considered as being representative of aerosols of 20 μm or less emitted by the patient.

The theoretical foundation of RTD analysis is based on an ideal pulse, or Dirac impulse function, where a known quantity of tracer is injected instantaneously at time $t = 0$. In practice, the tracer is introduced over a short but finite duration, creating a square or other non-instantaneous input function. To generate this input in the present simulations, the source term S in Equation (10) is specified as a unit impulse of tracer concentration over the volume of a single cough over the mouth of the patient and the time required to produce it. For convenience, this was taken as ~ 2 L over ~ 0.2 s, corresponding to an “optimal cough.” This choice of volume and duration is arbitrary and not required for RTD analysis and simply provides one way to approximate a finite pulse; any sufficiently short injection can serve the same purpose.

2.4 Computational setup

The numerical simulations were performed for two hospital isolation room designs specified by Thatiparti et al. [8, 9]. The first design considered was a design that promotes short-circuiting between the patient and exhaust vent by placing the exhaust vent closer to the patient which can be seen in Figure 3a (referred to as the short-circuiting design or SCD). A normal design (NMD) was also considered where the exhaust vent is located across the room from the patient as shown in Figure 3b, expected to lead to greater exposure of the HCW to the air or tracer expelled by the patient. Both SCD and NMD layouts were run with linear and square inlet (Figure 3) flow velocities such that their combined flow rate corresponded to 6 ACH and 12 ACH.

Boundary conditions and material properties of surfaces in the room were assigned following [8, 9] and are summarized in Table 1, Table 2 and Table 3.

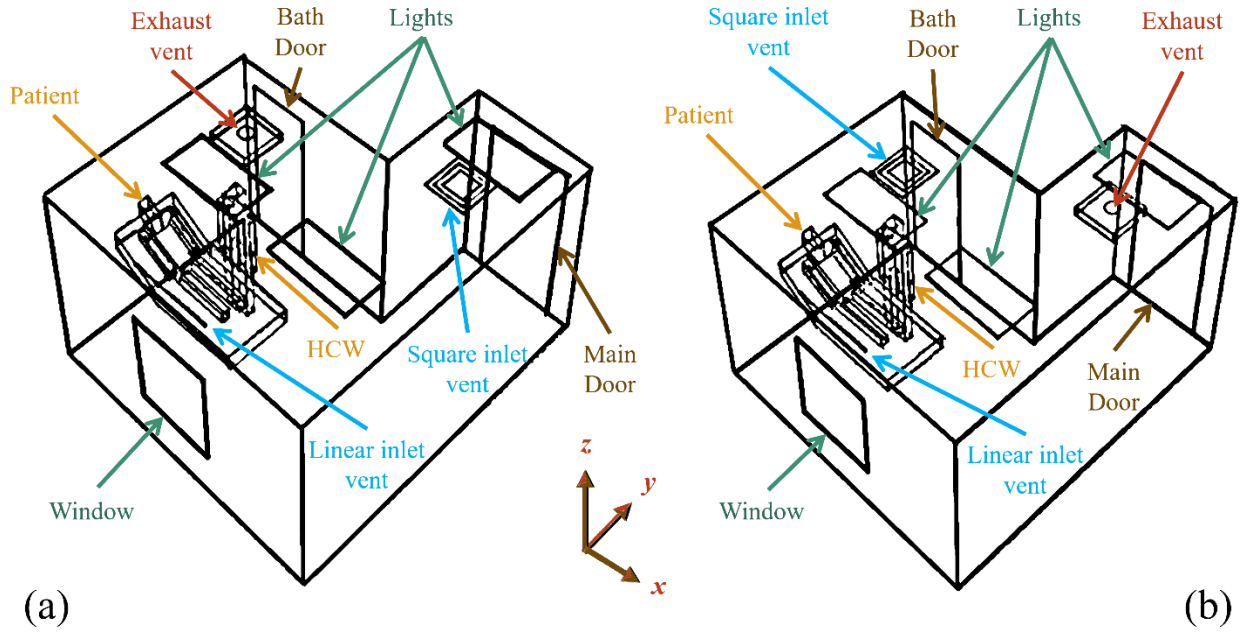


Fig. 3 Wireframe of two hospital isolation room geometries, given by Thatiparti et al. [8, 9], considered in the present CFD analysis: a design that promotes channeling between the patient and exhaust vent shown in (a) (i.e. the SCD room configuration) and a standard exhaust vent design in (b) (i.e. the NMD room configuration).

Table 1 Material properties of surfaces.

Surface	Material	Conductivity [W m ⁻¹ K ⁻¹]	Specific Heat [J kg ⁻¹ K ⁻¹]	Density [kg m ⁻³]
Window	Window glass	0.96	840	2500
Patient/HCW	Human skin	0.206	3558	1027
Overhead lights	Plastic	1.005	1670	1250
Doors	Wood	0.173	2310	700
Room walls	Dry wall	3.1	1090	600
Patient bed	Cotton fabric	0.043	1162	1540
Floor	Vinyl flooring	0.28	900	1450

Table 2 Momentum and temperature equations boundary conditions (BC). All modeled surfaces are noslip with a specified temperature as listed. Zero total (advective plus diffusive) normal flux was assigned for the scalar mass fraction at all of the surfaces listed, except at the exhaust (return housing) where only the diffusive normal flux was prescribed as zero.

Surface	Temperature [K]	Material
Patient	312	Cotton
Patient Head	312	Skin
HCW	309	Cotton
HCW Head	309	Skin
Bed	294	Cotton
Lights	294	Plastic
Walls	294	Dry Wall
Window	294	Glass
Floor	294	Vinyl Flooring
Doors	294	Wood
Ceiling	294	Dry Wall
Supply/Return Housing	294	Aluminum

Table 3 Inflow boundary conditions (BC). Note that both linear and square air supplies direct part the air flow at 45 degree angles from the ceiling. See Thatiparti et al. [8, 9] for more details about the geometry.

ACH	Surface	Mass flow rate [kg/s]
6	Linear Supply	0.0462
6	Square Supply	0.0377
12	Linear Supply	0.0923
12	Square Supply	0.0755

Meshing of the computational domain was carried out using the cut-cell algorithm [27], a tessellation-based Cartesian method, chosen for its ability to rapidly generate good quality meshes for simple geometries (e.g. see Figure 4). Both room configurations maintain nearly the same number of mesh elements at around 3 million, higher than the ~2.8 million element (finest) mesh reported in [8, 9].

The mesh size of 3 million was chosen after a mesh independence study of the NMD at 12 ACH case starting from a 1.5 million cell mesh. The procedure for increasing the mesh resolution was a simple halving of mesh size near inflows, outflows, and heat sources. The cell growth rate remained fixed, so the influence of this refinement extends beyond just the surface being refined. This refinement resulted in the 3 million cell mesh, whose parameters were used for the meshing of the SCD cases. Maximum skewness of mesh elements was kept to 0.7 (following recommendations in the literature, e.g. [28]), aiding the stability and convergence of the solution.

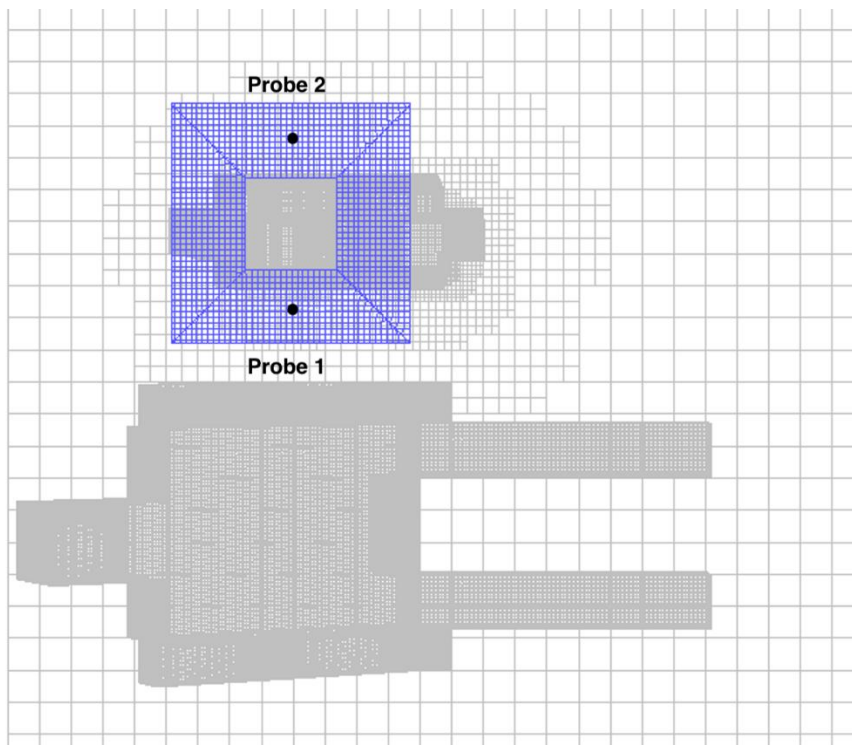


Fig. 4 Plan view of the mesh wireframe of the NMD isolation room setup showing the location of Probe 1 and Probe 2, which are vertical lines in front of and behind the HCW used to sample velocity magnitude and temperature profiles in Figure 5 as part of a mesh independence study.

The QUICK (Quadratic Upstream Interpolation for Convective Kinetics) scheme was used to discretize the momentum and temperature (energy) equations in (7) and (9), respectively, and the

turbulence transition SST model transport equations (not shown). These equations were integrated in time arriving at a steady state solution for the flow and temperature distribution throughout the room. The corresponding pressure was obtained via a staggered-grid pressure interpolation scheme, similar to classical schemes described in [29], ensuring satisfaction of the mass conservation equation in (6). Finally, the tracer transport equation in (10) was discretized via a 2nd order implicit method in time and a 1st order upwind method in space to obtain time dependent solutions.

The Reynolds-averaged flow was initially solved in the absence of temperature (compressible) effects. Once the homogeneous flow was obtained, the temperature equation in (9) was activated allowing for secondary flow characteristics (such as natural convection) to be incorporated into the solution obtained from the computation of the primary flow without temperature effects. This improved the rate of convergence towards a steady solution of the system. With the coupled momentum and temperature equations solved, the tracer transport equation in (10) (with source term, representing the release of patient-emitted tracer or air over the mouth of the patient, active for the first 0.2 seconds only as described earlier in sub-section 2.3), was run to track the tracer concentration throughout the room and over time.

The time step for the tracer transport equation in (10) was chosen such that the Courant-Friedrichs-Levy (CFL) number [29] based on ACH was kept roughly constant for each case, to allow consistent comparison between the cases with different ACH. Simulations at 6 ACH and 12 ACH (following [5, 8, 9]) for normal and short-circuiting room designs were performed, yielding a total of 4 simulations. The time step for the 6 ACH and 12 ACH simulations was 0.125 s and 0.0625 s, respectively.

2.5 Mesh sensitivity

Mesh sensitivity was considered by probing the flow field velocity magnitude and temperature along the vertical direction near the primary inflow for both the mesh described earlier (~3.0 million cells) and

a coarser mesh (~ 1.5 million cells) in the 12 ACH NMD case. A similar approach to test mesh independence was taken in [30] in their CFD analysis of flow in an intensive care unit. The locations of the vertical lines from which velocity magnitude and temperature are sampled are shown in Figure 4, denoted as Probe 1 and Probe 2. The resulting profiles are shown in Figure 5. The Probe 1 samples, collected along a vertical line between the HCW and the patient, exhibit sharp gradients in velocity and temperature near surfaces, such as the patient bed (at 0.9 m) and towards the top of the HCW (between $z = 1.5$ m and 1.7 m), which are indicative of thermal plumes. Both probes captured a jet from the air supply (inflow) near the ceiling (at $z = 2.5$ m).

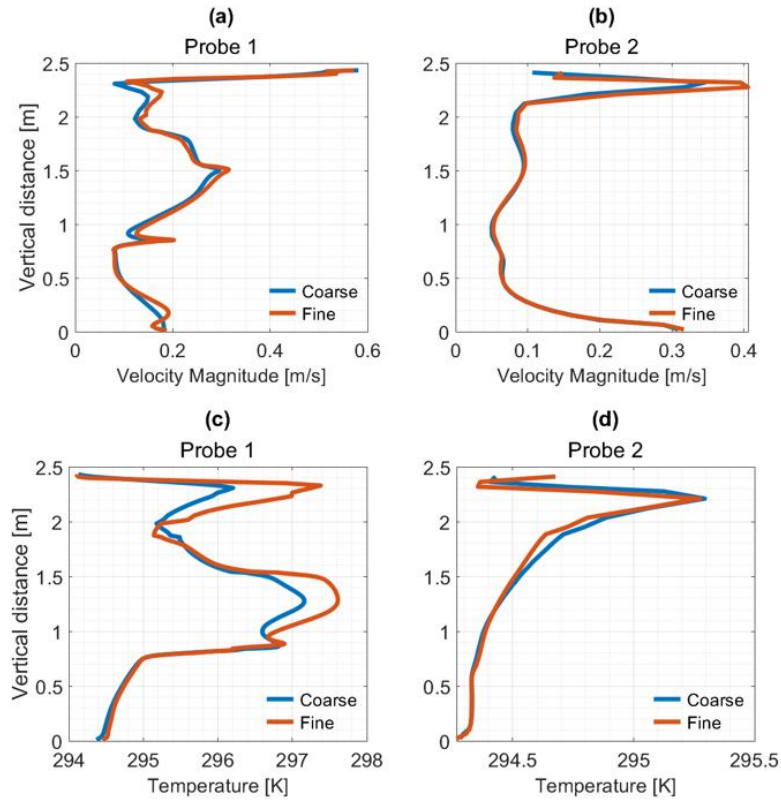


Fig. 5 Vertical profiles of velocity magnitude and temperature in the NMD 12 ACH room configuration case at the probe locations specified by Figure 4. Velocity magnitude and temperature profiles for Probe 1, subplots (a) and (c) respectively, and Probe 2, subplots (b) and (d) respectively, are shown for two different mesh fidelities referred to as coarse and fine.

The velocity magnitude and temperature profiles at each probe showed a root mean square difference between the coarse and fine meshes, ε_{rms} , of less than 10% and 1%, respectively, where ε_{rms} is defined as

$$\varepsilon_{\text{rms}} = \sqrt{\frac{1}{N} \sum_{i=1}^N \frac{(\phi_{\text{fine},i} - \phi_{\text{coarse},i})^2}{\phi_{\text{fine},i}^2}} \quad (12)$$

[30, 31], suggesting sufficient mesh resolution with the fine mesh. In Equation (12), ϕ is either velocity magnitude or temperature and N is the number of vertical sampling points of each probe.

2.6 Consistency checks

The HCW exposure to patient-emitted air (i.e. the tracer) analyzed further below in sub-section 3.4 is consistent with the exposure results of the benchmark study by Memarzadeh and Xu [5], suggesting a qualitatively valid computational setup.

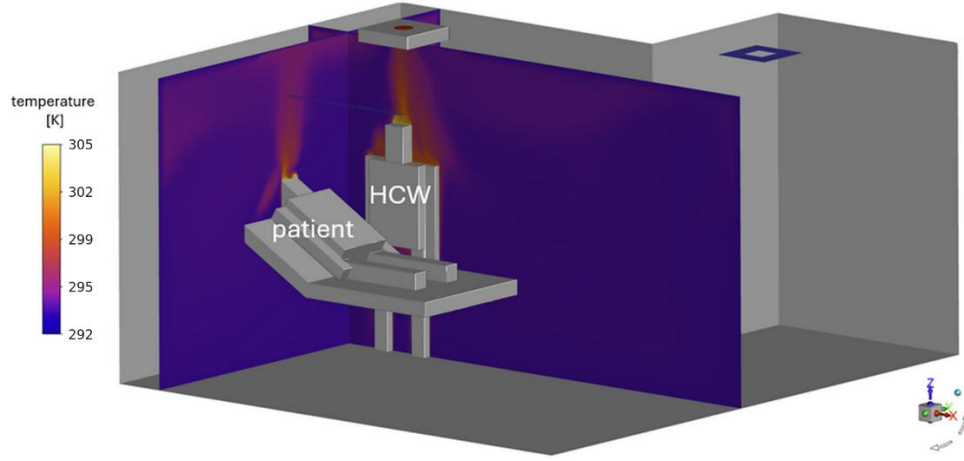
Additional consistency checks were made in terms of the thermal plumes associated with the patient and HCW. Figures 6a and 6b show, respectively, the temperature field and vertical velocity contours in the SCD at 12 ACH case, highlighting the buoyant thermal plumes generated above both the patient and the HCW. In Figure 6a, the elevated surface temperatures prescribed for the patient (312 K) and HCW (309 K) relative to the ambient room air (294 K) give rise to clear regions of warmed air above the head and torso. These warmer air regions coincide with upward motions visible in Figure 6b, where coherent plumes extend vertically towards the exhaust.

The area-averaged convective heat fluxes over the modeled head surfaces were approximately 102 W m^{-2} for the patient and 95 W m^{-2} for the HCW. The corresponding convective heat-release rate was calculated from $\dot{Q}_c = q_c'' A_h$, where q_c'' is the area-averaged convective heat flux and A_h is the exposed head surface area. Using $A_h = 0.37 \text{ m}^2$ gives $\dot{Q}_{c,\text{patient}} = 37.74 \text{ W}$, and $\dot{Q}_{c,\text{HCW}} = 35.15 \text{ W}$.

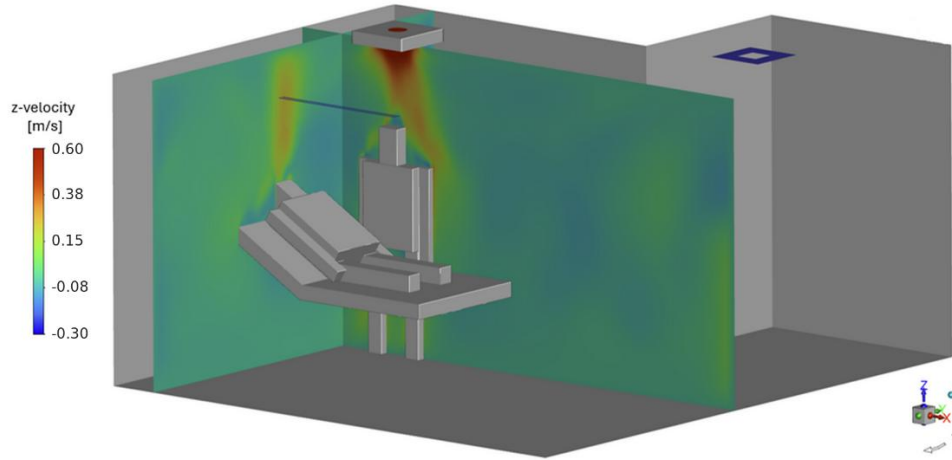
The buoyancy flux associated with each heat source was estimated as

$$B = \frac{g\dot{Q}_c}{\rho_\infty c_p T_\infty} \quad (13)$$

where $g = 9.81 \text{ m s}^{-2}$, $\rho_\infty = 1.204 \text{ kg m}^{-3}$, $c_p = 1006 \text{ J kg}^{-1} \text{ K}^{-1}$, and $T_\infty = 294 \text{ K}$. The resulting buoyancy fluxes are $B_{\text{patient}} = 1.04 \times 10^{-3} \text{ m}^4 \text{ s}^{-3}$, and $B_{\text{HCW}} = 9.68 \times 10^{-4} \text{ m}^4 \text{ s}^{-3}$.



(a)



(b)

Fig. 6 (a) Temperature and (b) vertical (z) velocity contours near the patient and HCW for the SCD 12 ACH case.

For an axisymmetric pure plume, the Morton-Taylor-Turner top-hat formulation [32] gives the characteristic vertical plume velocity as

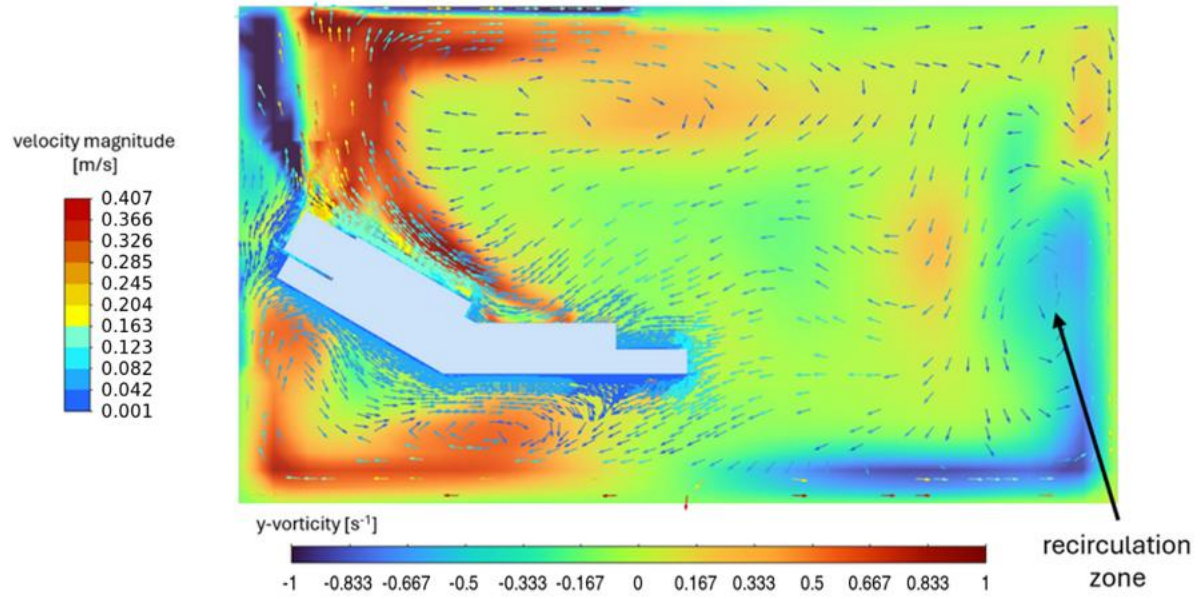
$$w(z) = \left(\frac{3}{4\pi}\right)^{1/3} \left(\frac{5}{6\alpha}\right)^{2/3} B^{1/3} z^{-1/3} \quad (14)$$

where α is the entrainment coefficient and z is the distance above the effective plume origin. Taking $\alpha = 0.10$ [32], assuming the head to be the approximate plume origin, and evaluating the expression at $z = 1$ m gives $w_{\text{patient}}(1 \text{ m}) \approx 0.258 \text{ m s}^{-1}$, and $w_{\text{HCW}}(1 \text{ m}) \approx 0.252 \text{ m s}^{-1}$. These analytical estimates are of the same order as the approximately $0.3\text{--}0.4 \text{ m s}^{-1}$ vertical velocities suggested by the CFD contours (Figure 6b). Exact agreement is not expected because the classical plume expression assumes an isolated point-source plume in a quiescent and uniform environment. In contrast, the CFD plumes originate from finite heated surfaces, interact with the ventilation flow, and are affected by walls, the bed, and the nearby exhaust. Nevertheless, these calculations confirm that the imposed thermal boundary conditions generate realistic buoyant plumes in the CFD simulations consistent with theoretical scaling, which is an important check on the validity of the flow and tracer transport predictions presented further below.

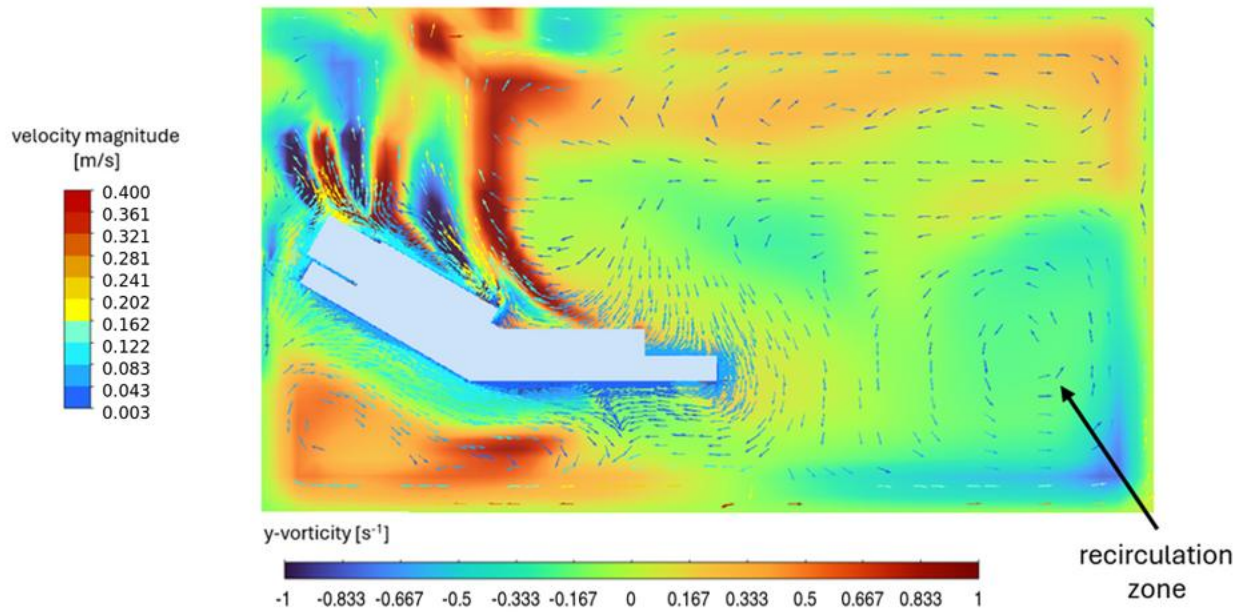
3 Results

3.1 Visualizations of flow structures: Recirculation zones and short-circuiting

Figure 7 presents velocity vectors with contours of y -vorticity in the vertical plane intersecting the patient and bed for the SCD (panel a) and NMD (panel b) layouts at 12 ACH. In the SCD case, where the exhaust is located above the patient and HCW (see Fig. 3a), the upward jet produced by the buoyant plume over the patient is well aligned with the outlet. The y -vorticity contours above the patient are concentrated, indicating strong shear associated with a coherent plume feeding directly into the exhaust. Recirculation or dead zones are present but confined to the far-right side near the right wall; one of these zones is denoted by the arrow.



(a)



(b)

Fig. 7 Velocity vectors color coded by magnitude and y-vorticity contours over the x-z plane along the middle of the patient and bed for (a) the SCD and (b) the NMD room configurations at 12 ACH.

In the NMD case (Fig. 7b), where the exhaust is displaced away from the patient (see Fig. 3b), the buoyant updraft from the patient is less directly coupled to the outlet. The y-vorticity field above the patient is still strong, but characterized by smaller spatial scales, reflecting plume interaction with flow structures near the room ceiling. The arrow in Fig. 7b points to a large recirculation zone along the far-right side of the room that is noticeably broader than in the SCD case (Fig. 7a). Alignment of the patient plume with the exhaust in the SCD case is accompanied by a more direct removal pathway and a smaller far-wall recirculation region. In the NMD case, poorer plume–exhaust alignment is accompanied by broader room-scale recirculation, providing a plausible mechanism for the greater tracer retention subsequently observed below.

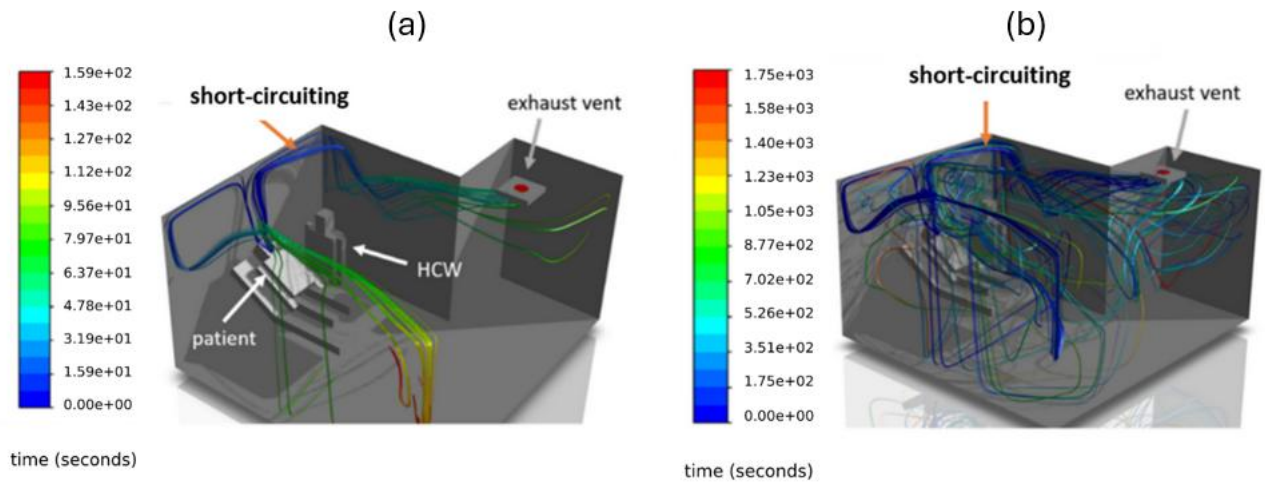


Fig. 8 Pathlines traced out by a fixed number of fluid parcels released from the mouth of the patient in the NMD 12 ACH case. Panels (a) and (b) show the pathlines traced out by the fluid parcels at 159 s and 1750 s, respectively, after the particles were released. The pathlines are color-coded by time from when the parcels were released.

Figure 8 further illustrates the flow structure in the NMD 12 ACH case using pathlines originating near the patient's mouth. The pathlines show the upward transport of patient-emitted air, its deflection

near the ceiling, and its subsequent interaction with the bulk room flow. Some trajectories eventually reach the exhaust through the upper portion of the room, whereas others undergo substantially longer excursions through the room. These trajectories illustrate the coexistence of relatively direct removal pathways and room-scale recirculation. The RTD analysis presented below quantifies the combined effects of early exhaust breakthrough and longer tracer retention for all simulated cases. Comparisons are made between the CFD simulated cases and the theoretical perfectly mixed model in which by definition short-circuiting and dead zones do not occur.

Figure 9 presents the spatial evolution of a fixed tracer-concentration isosurface for the SCD and NMD configurations at 12 ACH. At $t = 5$ s after the tracer has been released (Figs. 9a,b), the tracer plume in the SCD configuration has already formed a direct upward connection between the patient and the nearby exhaust. In the NMD configuration, the corresponding plume remains concentrated above the patient and is separated from the more distant exhaust. Thus, despite having the same ACH, the two exhaust placements establish distinctly different early transport pathways: prompt source capture in the SCD configuration versus delayed removal and greater opportunity for room-scale mixing in the NMD configuration. The resulting difference in early breakthrough is quantified below using the RTD-derived t_{10} .

At $t = 35$ s (Figs. 9c,d), the NMD tracer isosurface is substantially more room-filling and extends farther into the lower occupied portion of the room. The SCD isosurface also spreads beyond the direct patient-to-exhaust pathway, demonstrating that not all of the discharged tracer is captured immediately, but its spatial extent remains smaller than in the NMD case. By $t = 85$ s (Figs. 9e,f), the tracer concentration in the SCD case has fallen below the plotted isovalue throughout the room, whereas the NMD case retains a coherent wall-adjacent tracer pocket. This persistent pocket is consistent with tracer retention in recirculating flow followed by gradual release through turbulent diffusion. Because Figure 9

displays a single concentration isovalue, the absence of a visible surface does not imply complete tracer removal; rather, it indicates that the local tracer concentration has fallen below the selected threshold.

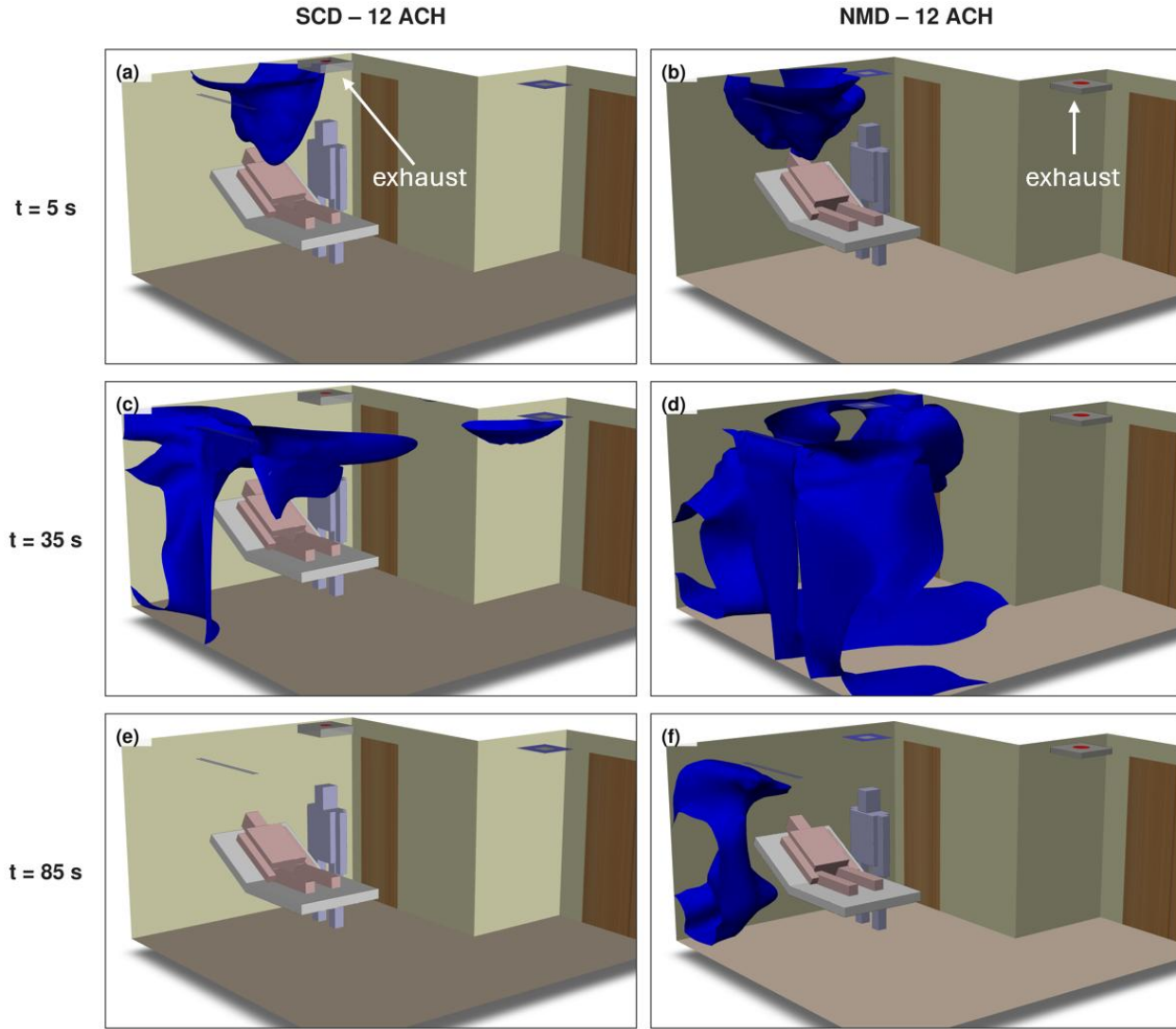


Fig. 9 Evolution of a patient-discharged-air isosurface for the SCD and NMD configurations at 12 ACH.

The same tracer-mass-fraction isovalue, $8 \times 10^{-5} \text{ kg}_{\text{tracer}} \text{ kg}_{\text{air}}^{-1}$, is used in all panels.

3.2 RTD and bulk (room-averaged) tracer concentrations

Looking at Figure 10, RTD time series (defined in Eqn. (1) based on the average concentration at the exhaust) show marked differences between the SCD and the NMD. In the SCD cases, a sharp early peak appears, especially at 12 ACH, consistent with strong short-circuiting pathways connecting the patient's mouth to the exhaust. In the NMD cases, the early peak is muted, reflecting weaker short-circuiting and stronger recirculation. Secondary peaks in the SCD exhaust histories around 0.9 and 1.5 min may represent tracer arriving through multiple transport pathways, including delayed release from recirculating regions by turbulent diffusion. The long late-time tails are similarly consistent with gradual tracer release from regions that are weakly connected to the exhaust. At late times, the RTD decay trends approach the perfectly mixed model prediction, suggesting that the influence of the initial source-to-exhaust pathway diminishes as the remaining tracer becomes more broadly dispersed throughout the room.

The early peaks and long tails of the RTD in Figure 10 are quantified by the RTD indices summarized in Tables 4 and 5. At 12 ACH, the SCD breakthrough time is $t_{10} = 0.140$ min, compared with 1.55 min for NMD. Thus, breakthrough occurs approximately 11 times earlier in the SCD case, consistent with a stronger patient-to-exhaust short-circuiting pathway. At 6 ACH, the contrast remains appreciable but is smaller: $t_{10} = 0.507$ min for SCD and 1.35 min for NMD. At later times, t_{90} also separates the 12-ACH configurations, with values of 8.27 min for SCD and 12.92 min for NMD, whereas the two 6-ACH cases have relatively similar values of 20.97 and 22.09 min, respectively. Comparison with the PMA values in Table 4 shows that these differences reflect more than a difference in the degree of short-circuiting. At 12 ACH the PMA breakthrough time is 0.523 min, so the SCD case breaks through approximately 3.7 times earlier than a perfectly mixed room while the NMD case breaks through approximately 3.0 times later. The same contrast appears at 6 ACH, where the PMA value is

1.05 min against 0.507 min for SCD and 1.35 min for NMD. The late-time index behaves similarly: at 12 ACH, $t_{90} = 8.27$ min for SCD and 12.92 min for NMD, compared with 11.43 min for the PMA. The two exhaust placements therefore fall on opposite sides of the perfectly mixed response rather than differing only in magnitude.

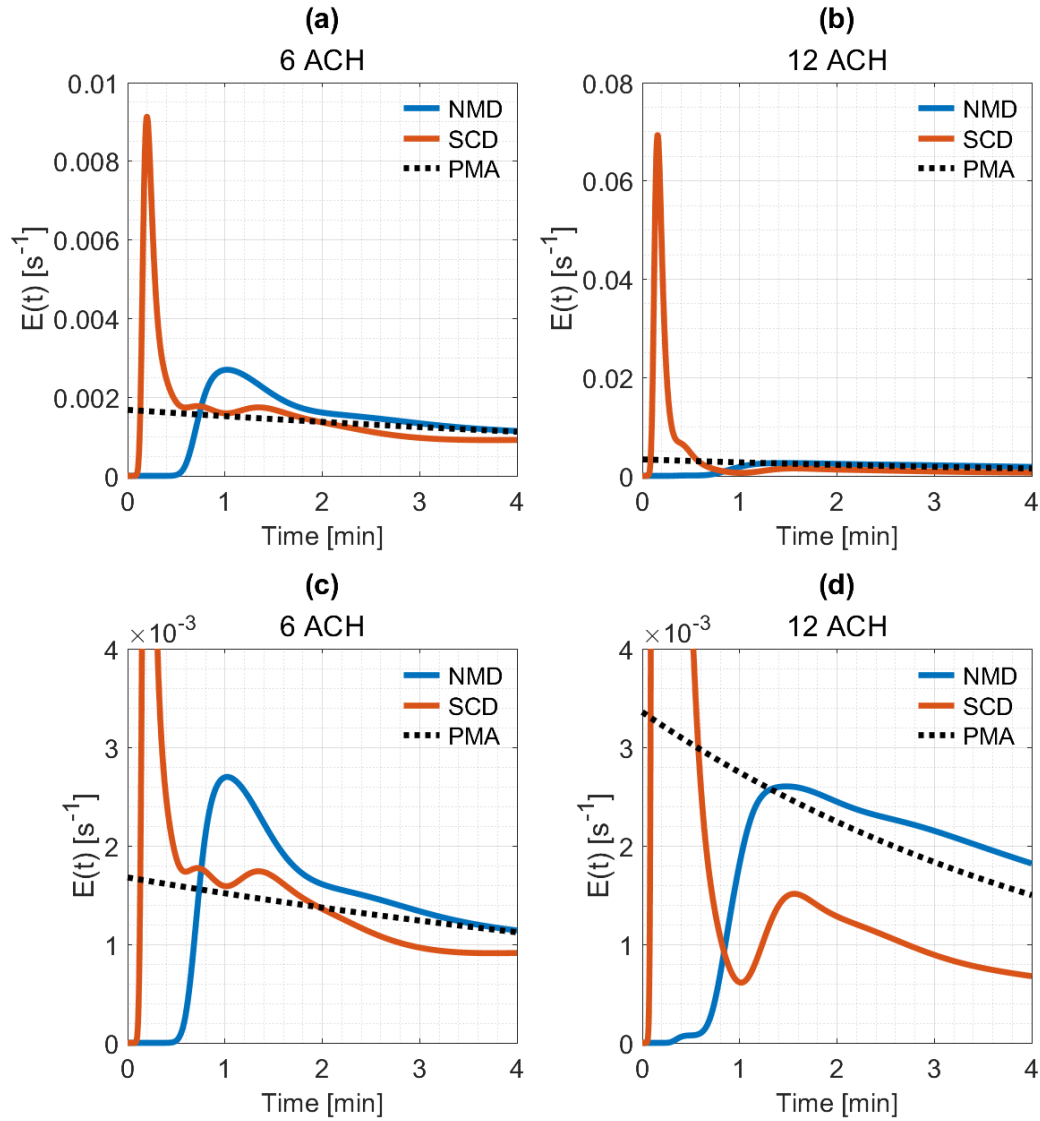


Fig. 10 RTD in the NMD and SCD ACH = 6 and ACH = 12 cases and in theoretical model (based on the perfectly mixed assumption (PMA)). Panels (c) and (d) show zoomed-in versions of (a) and (b), respectively.

The apparent dead-volume fraction, $\phi_d = 1 - \tau_{CFD}/\tau_{theor}$ (listed in Table 5 for all cases), reinforces these interpretations. The SCD 12 ACH case has $\phi_d = 0.468$, corresponding to an apparent bypass fraction of approximately 47% relative to the perfectly mixed residence time. In contrast, the NMD 12 ACH case has $\phi_d = -0.255$ because $\tau_{CFD} > \tau_{theor}$. This negative value does not represent a negative physical dead volume; rather, it indicates delayed tracer retention relative to the perfectly mixed model. At 6 ACH, the values are much closer to zero (0.096 for SCD and 0.022 for NMD), indicating that exhaust placement has a substantially smaller influence on the mean removal timescale at the lower ventilation rate.

Table 4 Dimensional time indices in minutes.

Case	τ_{theor} [min]	τ_{CFD} [min]	t_{10} [min]	t_{90} [min]	$t_{90} - t_{10}$ [min]
NMD 6 ACH	9.93	9.70	1.35	22.09	20.73
SCD 6 ACH	9.93	8.97	0.507	20.97	20.47
PMA 6 ACH	9.93	9.93	1.05	22.86	21.81
NMD 12 ACH	4.96	6.23	1.55	12.92	11.37
SCD 12 ACH	4.96	2.64	0.140	8.27	8.13
PMA 12 ACH	4.96	4.96	0.523	11.43	10.91

Table 5 Dimensionless indices.

Case	$\phi_d = 1 - \tau_{CFD}/\tau_{theor}$	t_{10}/τ_{theor}	$W = (t_{90} - t_{10})/\tau_{theor}$	MI = t_{90}/t_{10}
NMD 6 ACH	0.022	0.136	2.09	16.33
SCD 6 ACH	0.096	0.0511	2.06	41.38
NMD 12 ACH	-0.255	0.311	2.29	8.36
SCD 12 ACH	0.468	0.0283	1.64	58.88
PMA 6/12 ACH	0	0.105	2.20	21.85

This conclusion is consistent with the bulk (room-averaged) tracer concentration time series in Figure 11. At 6 ACH, the SCD design does not appreciably reduce the average room concentration relative to the NMD design and the perfectly mixed model prediction throughout the simulation. At 12 ACH, however, the SCD does produce noticeably lower bulk concentrations than the NMD case, reflecting the former's effectiveness at sufficiently high flow strength.

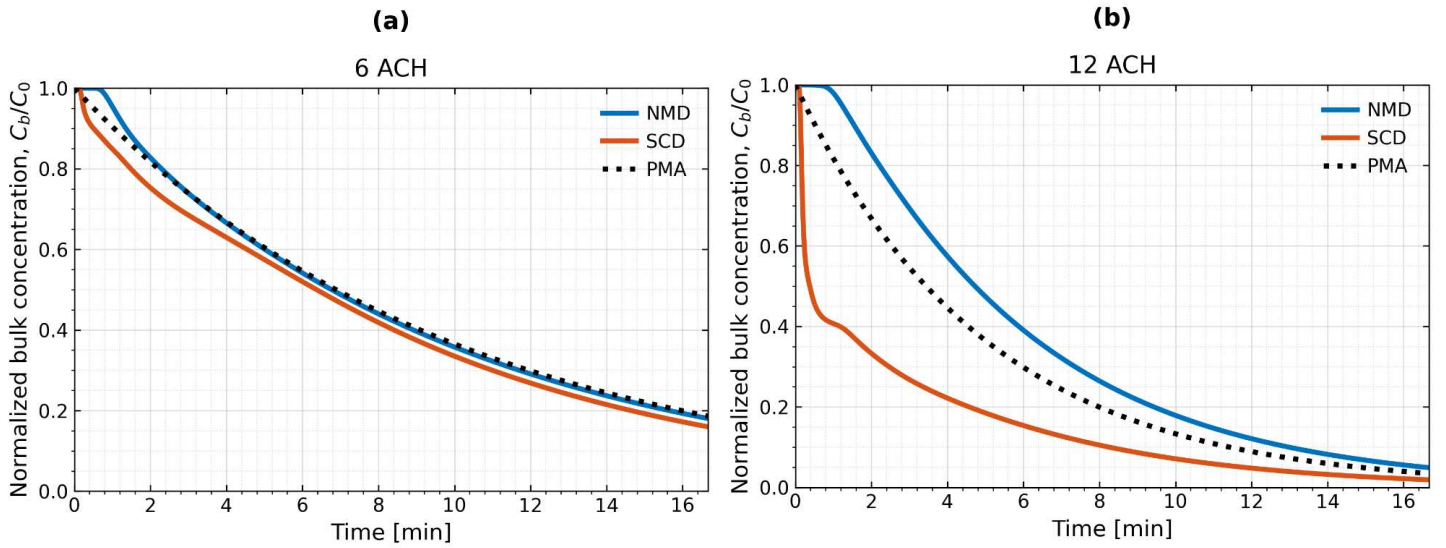


Fig. 11 Normalized bulk (room-averaged) concentration in (a) the NMD and SCD ACH = 6 and (b) ACH = 12 cases and in theoretical model (based on the perfectly mixed assumption (PMA)). The bulk concentration has been normalized by the initial room-averaged concentration.

A noticeable feature of the NMD bulk concentration curves is their initial flat period. In the NMD 12 ACH case (Fig. 11b), the bulk concentration remains essentially constant during the first minute, unlike the perfectly mixed model which begins decaying immediately. This lag arises because in the CFD simulations the tracer is released as a finite 0.2-s pulse at the patient's mouth and requires finite transport time to reach the exhaust, so the average room concentration does not begin to decrease until tracer starts leaving. In contrast, the SCD design significantly reduces the lag effect, while the perfectly mixed model assumes instantaneous uniform mixing throughout the entire room volume, so removal to the exhaust starts at $t = 0$. The lag effect causes the NMD 12 ACH curve to stay above that of the perfectly mixed model throughout the simulation. This lag is quantified by the breakthrough time, which is 1.55 min for NMD 12 ACH against 0.523 min for the PMA (Table 4). A similar behavior is observed in the NMD 6 ACH case (Fig. 11a); however, it is less pronounced because the initial flat period in bulk concentration occurs over a shorter interval.

Over time, the NMD bulk-concentration curves gradually approach the perfectly mixed predictions. The NMD 6 ACH case has τ_{CFD} close to τ_{theor} (see Table 4), whereas the longer mean residence time of the NMD 12 ACH case is consistent with its more pronounced initial delay and subsequent tracer retention.

3.3 Cumulative RTD

Recall that the shape of the cumulative RTD provides insight into whether the flow is advection-dominated or diffusion-dominated. For the flows considered, a steep rise in the cumulative RTD would indicate an advection-dominated flow induced by strong short-circuiting, whereas a milder rise would indicate a diffusion-dominated flow associated with recirculation and turbulent diffusion (see subsection 2.1 and Figure 2). The spread window, $t_{90} - t_{10}$, can be used as an index to help characterize the breadth of the cumulative RTD, and therefore the relative contributions of early breakthrough and late-time persistence.

Figure 12a shows that the cumulative RTDs for the NMD and SCD designs at 6 ACH closely follow the perfectly mixed model prediction. Both curves rise gradually and reach 90% removal only after approximately 21–22 min, consistent with diffusion-dominated persistence: $t_{90} = 22.09$ min for NMD and 20.97 min for SCD, against 22.86 min for the PMA (Table 4). The corresponding spread windows are $t_{90} - t_{10} = 20.73$ min for NMD and 20.47 min for SCD. The modest separation early in time reflects the earlier breakthrough for SCD, with $t_{10} = 0.507$ min compared with 1.35 min for NMD.

At 12 ACH (Fig. 12b), the cumulative RTD curves diverge strongly. SCD exhibits a much steeper early rise, characteristic of robust short-circuiting, with $t_{10} = 0.140$ min compared with 1.55 min for NMD. By later times, the SCD curve remains ahead, with t_{90} decreasing from 12.92 min for NMD to 8.27 min for SCD. Accordingly, the spread window narrows from 11.37 min for NMD to 8.13 min for

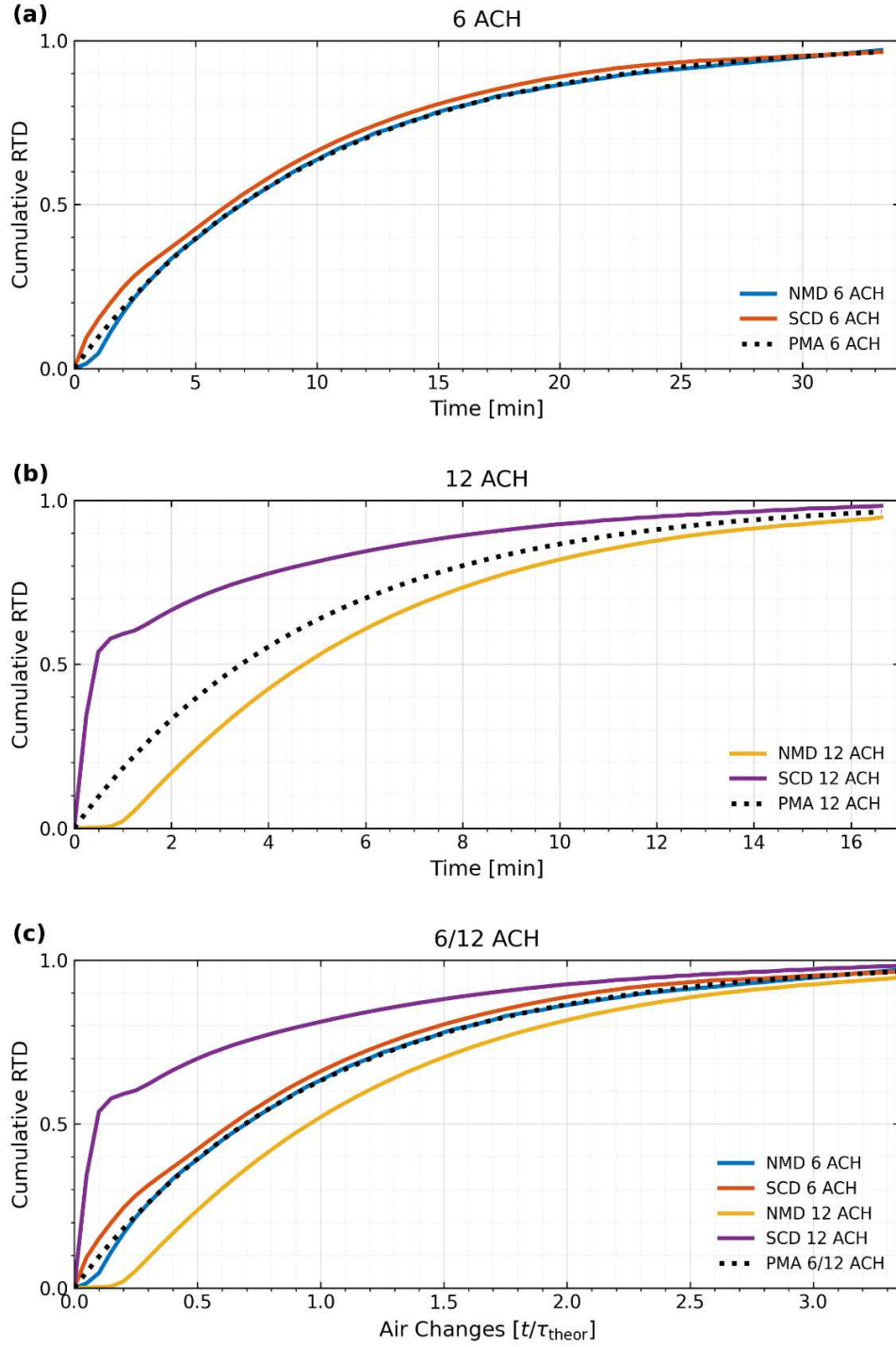


Fig. 12 Comparison of the CFD-computed cumulative RTDs with the theoretical model based on the perfectly mixed assumption (PMA) for (a) 6 ACH, (b) 12 ACH, and (c) all four cases plotted against normalized time, t/τ_{theor} .

SCD, indicating a more advection-dominated response under SCD at high ACH. The Morrill index (listed in Table 5) further emphasizes the difference: $MI = 58.88$ for SCD 12 ACH compared with 8.36 for NMD 12 ACH, against 21.85 under the PMA. This large difference is driven primarily by the very small t_{10} of the SCD case rather than by a more persistent late-time tail; in fact, both t_{90} and $t_{90} - t_{10}$ are smaller for SCD. The NMD 12 ACH case also exhibits the early-time lag identified previously in the bulk tracer concentration.

The PMA value of the Morrill index is $\ln(10)/\ln(10/9) = 21.85 \sim 22$, conventionally quoted as the complete-mixing limit, with $MI = 1$ denoting ideal plug flow [21, 23]. Both pairs of configurations fall on opposite sides of this value: $MI = 16.33$ for NMD and 41.38 for SCD at 6 ACH, and 8.36 and 58.88 at 12 ACH. Exhaust placement therefore determines whether the removal response is more or less plug-flow-like than complete mixing, while ventilation rate sets how far to either side the configuration moves.

Normalizing time by the theoretical mean residence time places all four cases on a common ventilation-timescale basis. As shown in Figure 12c, the NMD and SCD cases at 6 ACH remain close to one another and approximately follow the perfectly mixed model prediction. The NMD 12 ACH curve exhibits a more pronounced initial delay, followed by gradual convergence toward the perfectly mixed trend. In contrast, the SCD 12 ACH curve rises sharply at early normalized times and remains above the other curves, demonstrating rapid exhaust breakthrough produced by the direct patient-to-exhaust pathway.

These differences are summarized by the normalized spread, $(t_{90} - t_{10})/\tau_{theor}$, listed in Table 5. For a perfectly mixed system, $(t_{90} - t_{10})/\tau_{theor} = 2.20$. The NMD 6 ACH, SCD 6 ACH, and NMD 12 ACH cases have normalized spreads of 2.09, 2.06, and 2.29, respectively. These values bracket and

remain relatively close to the perfectly mixed benchmark. In contrast, the SCD 12 ACH case has the smallest normalized spread, 1.64, together with a very small normalized breakthrough time, $t_{10}/\tau_{theor} = 0.0283$, well below the PMA value of 0.105, whereas the NMD 12 ACH case breaks through late at 0.311 (Table 5). This combination in the SCD 12 ACH case indicates rapid initial breakthrough followed by a comparatively narrow removal response, consistent with transport being strongly influenced by organized advection through the short-circuiting pathway. At 6 ACH, the SCD pathway does not produce the same degree of separation from the NMD and perfectly mixed curves because turbulent dispersion and recirculation broaden the response. Thus, within the configurations studied, the influence of the SCD pathway is most pronounced when combined with the higher ventilation rate. It should be emphasized, however, that $(t_{90} - t_{10})/\tau_{theor}$ characterizes only the width of the cumulative RTD; curves with similar values of this index may still differ in their early-time shape and transport behavior.

3.4 HCW exposure

The HCW exposure histories are presented in Figure 13a. HCW exposure is defined as the cumulative time integral of the normalized patient-emitted tracer concentration averaged within 0.3 m of the HCW's head,

$$\mathcal{E}_H(t) = \int_0^t C_H^*(t') dt' \quad (15)$$

where C_H^* is the tracer concentration near the HCW normalized by the initial room-average tracer concentration. This definition follows that given by Memarzadeh and Xu [5].

Figure 13a shows that increasing ACH does not, by itself, determine the short-term exposure. Over approximately the first 2–5 min, the NMD 6 ACH, SCD 6 ACH, and NMD 12 ACH cases remain within the same broad exposure range, whereas the SCD 12 ACH case exhibits a distinctly lower exposure.

Thus, doubling the ventilation rate produces a pronounced short-term benefit only when it is combined

with the direct patient-to-exhaust pathway present in the SCD configuration. Increasing ACH without establishing this pathway, as in the NMD configuration, provides a much smaller short-term benefit.

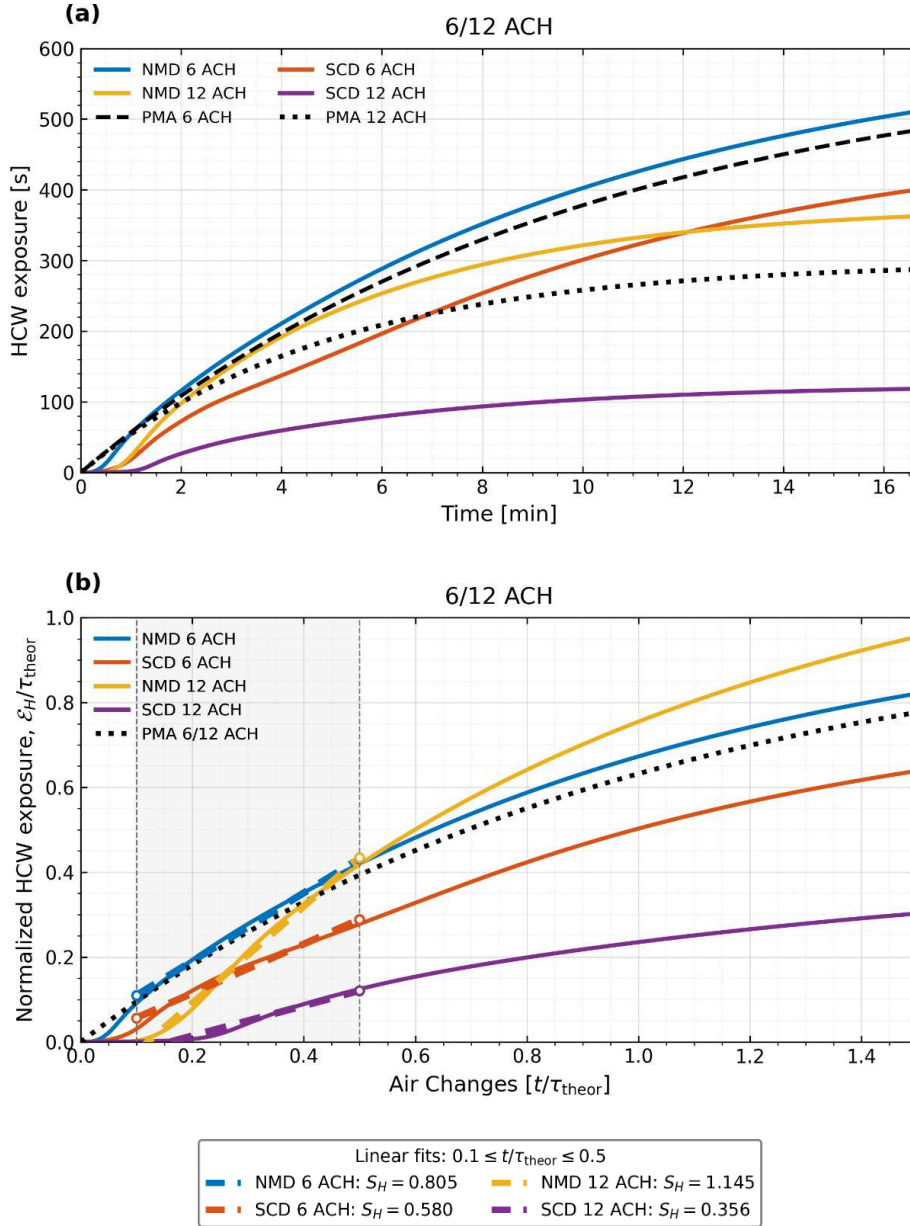


Fig. 13 (a) Normalized HCW exposure versus time and (b) normalized exposure scaled by τ_{theor} versus normalized time in simulations cases at 6 and 12 ACH. Additionally, HCW exposure under the perfectly mixed assumption (PMA) is shown. Slopes of the linear least-squares fits in panel (b) correspond to the normalized exposure rate, S_H , defined in Eqn. (16).

This behavior is qualitatively consistent with the “path principle” described by Memarzadeh and Xu [5]. In their simulations, increasing ACH had relatively little influence on HCW exposure when the room layout interrupted the source-to-exhaust transport pathway, whereas increasing ACH reduced exposure when that pathway was preserved. Similarly, the present results show that the largest early-time reduction occurs for the SCD 12 ACH case, in which the exhaust location and higher ventilation rate act together to rapidly transport patient-emitted air away from the HCW. This qualitative agreement supports the physical interpretation of the present simulations, although differences in geometry, source specification, and exposure definition prevent a direct quantitative comparison between the two studies.

Figure 13b places the four exposure histories on the common normalized timescale t/τ_{theor} . The linear least-square fits over $0.1 \leq t/\tau_{theor} \leq 0.5$ (dashed line segments in Fig. 13b) define the mean normalized HCW exposure rate,

$$S_H = \frac{d(\mathcal{E}_H/\tau_{theor})}{d(t/\tau_{theor})} \quad (16)$$

The corresponding values are 0.805, 0.580, 1.145, and 0.356 for NMD 6 ACH, SCD 6 ACH, NMD 12 ACH, and SCD 12 ACH, respectively. The corresponding PMA value, obtained by applying the same least-squares procedure to the analytical perfectly mixed exposure curve, is 0.744 (Table 6). Both NMD cases exceed this value and both SCD cases fall below it. In particular, the NMD 12 ACH case accumulates exposure more rapidly than a perfectly mixed room would, and more rapidly than its own 6 ACH counterpart, despite operating at twice the ventilation rate. Exhaust placement, rather than ventilation rate, determines on which side of the perfectly mixed response the short-term exposure accumulation falls. These results further demonstrate that ACH alone is not an adequate predictor of HCW exposure; its effect depends on how the ventilation flow interacts with the room layout and the patient-to-exhaust transport pathway.

The long-term exposure histories (in terms of asymptotic exposures) reveal benefits of increasing ACH that are less apparent during the first several minutes. The asymptotic exposures are 617.62 and 377.79 s for the NMD 6 and 12 ACH cases, respectively, and 491.53 and 124.12 s for the corresponding SCD cases (Table 6). Increasing ACH from 6 to 12 therefore reduces the long-term exposure by approximately 39% for NMD and 75% for SCD. At a fixed ACH, changing from NMD to SCD reduces the long-term exposure by approximately 20% at 6 ACH and 67% at 12 ACH. The influence of exhaust placement is therefore considerably stronger at the higher ventilation rate.

For a perfectly mixed room, the asymptotic exposure is equal to τ_{theor} , giving approximately 595.6 s at 6 ACH and 297.8 s at 12 ACH. Normalizing the asymptotic exposures of the CFD cases by these benchmarks gives 1.04 for NMD and 0.83 for SCD at 6 ACH, and 1.27 and 0.42 at 12 ACH (Table 6). At 6 ACH both configurations remain within approximately 20% of the perfectly mixed prediction, consistent with the corresponding cumulative RTDs being close to the perfectly mixed response. At 12 ACH, however, the normalized NMD exposure exceeds the perfectly mixed prediction while the SCD exposure falls well below it (Table 6). The perfectly mixed assumption therefore obscures the strong influence of airflow topology at the higher ventilation rate: delayed transport and recirculation increase exposure in the NMD configuration, while the organized patient-to-exhaust pathway substantially reduces exposure in the SCD configuration.

3.5 HCW exposure and RTD metrics

A comparison between HCW exposure and cumulative RTD behavior is natural because both quantities are constructed from time integrals of concentration. HCW exposure is the time integral of the concentration sampled near the HCW (recall Eqn. (15)), whereas the cumulative RTD is obtained by integrating the tracer response measured at the exhaust (recall Eqn. (5)). The two quantities are not equivalent: one characterizes local accumulation at the HCW and the other characterizes the fraction of

tracer that has exited the room. Nevertheless, both reflect the same underlying transport processes, including direct advection, turbulent dispersion, recirculation, and late-time retention. It is therefore useful to examine whether the HCW exposure behavior observed in the four simulations can be interpreted in relation to the cumulative RTD metrics.

Figure 14a and Table 6 compare long-term HCW exposure with the CFD mean residence time, τ_{CFD} . The cases with longer mean residence times generally produce greater accumulated HCW exposure. The NMD 6 ACH and SCD 6 ACH cases have $\tau_{CFD} = 9.70$ and 8.97 min and long-term HCW exposures of 617.62 and 491.53 s, respectively. The 12-ACH cases exhibit a greater separation: τ_{CFD} decreases from 6.23 min for NMD to 2.64 min for SCD, while long-term exposure decreases from 377.79 to 124.12 s. The descriptive linear fit gives $R^2 = 0.974$. The PMA cases, shown as open diamonds in Figure 14a, provide an independent check on this trend. Their values were not used in constructing the fit, yet both lie close to it, within 1.5% at 6 ACH and 6.5% at 12 ACH, which is comparable to the scatter of the CFD cases themselves. The analytical perfectly mixed solution therefore falls on a relationship derived entirely from the CFD results.

Within the four CFD cases, the trend in Figure 14a indicates that long-term exposure is closely associated with the overall time required to remove patient-emitted air. It does not imply that τ_{CFD} uniquely determines local HCW exposure, because exposure also depends on the spatial relationships among the patient, HCW, supply, and exhaust.

Short-term behavior is considered in Figure 14b and Table 6 by comparing short-term HCW exposure rate, S_H (defined earlier in Eqn. (16)), with the normalized cumulative-RTD spread, $W = (t_{90} - t_{10})/\tau_{theor}$. Recall that the normalized spread characterizes the breadth of the exhaust-removal response, while S_H characterizes the rate at which exposure accumulates near the HCW over $0.1 \leq t/\tau_{theor} \leq 0.5$. The descriptive fit gives $R^2 = 0.842$, indicating that these two quantities vary

systematically across the four cases. The SCD 12 ACH case has both the smallest normalized spread and the smallest exposure rate. Together with its very small t_{10}/τ_{theor} , this behavior is consistent with rapid exhaust breakthrough and organized advection through the patient-to-exhaust pathway, which limits accumulation near the HCW. The other three cases have broader cumulative RTDs and larger values of S_H , reflecting a greater overall role of turbulent dispersion and recirculation in the tracer-removal response.

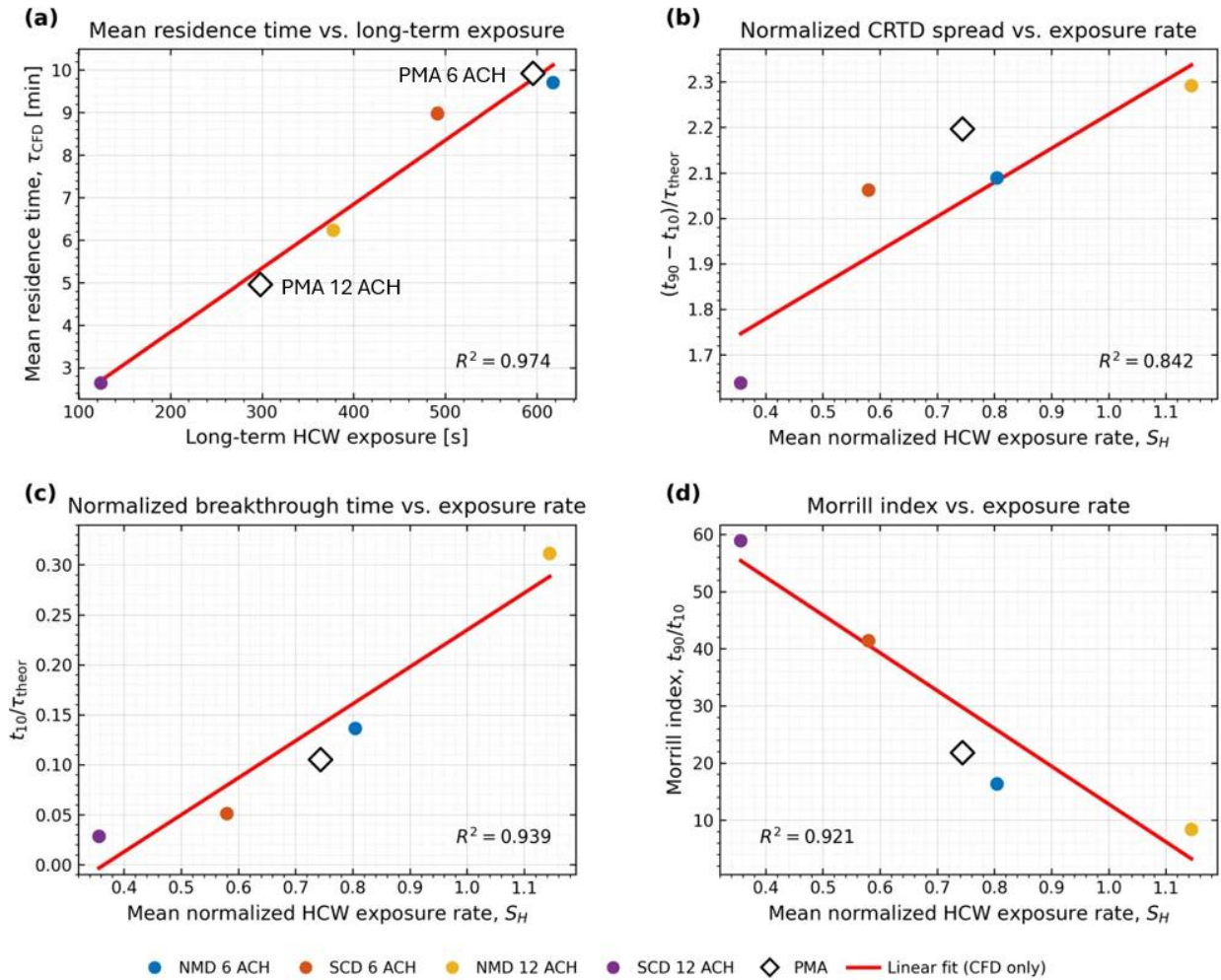


Fig. 14 Relationships between HCW exposure measures and cumulative RTD metrics for the four simulated cases: (a) long-term HCW exposure versus τ_{CFD} ; (b) S_H versus normalized cumulative-RTD spread, $(t_{90} - t_{10})/\tau_{theor}$; (c) S_H versus normalized breakthrough time, t_{10}/τ_{theor} ; and (d) S_H versus

the Morrill index, $MI = t_{90}/t_{10}$. Red lines represent linear least-squares fits included as descriptive guides for the four cases and not as general predictive correlations. Open diamonds denote the perfectly mixed assumption (PMA). In panel (a) the PMA values differ between 6 and 12 ACH and are labeled accordingly; in panels (b)–(d) the metrics are normalized by τ_{theor} so the PMA value is identical at both ventilation rates and a single symbol is shown. Linear fits were obtained from the four CFD cases only and exclude the PMA points.

Table 6 Short-term and long-term HCW exposure and various time indices.

Case	τ_{CFD} [min]	$\frac{t_{10}}{\tau_{theor}}$	W $= \frac{(t_{90} - t_{10})}{\tau_{theor}}$	MI $= \frac{t_{90}}{t_{10}}$	Long-term HCW exposure [s]	Long-term HCW exposure / τ_{theor}	Short-term HCW exposure rate, S_H
NMD 6 ACH	9.70	0.136	2.09	16.33	617.62	1.037	0.805
SCD 6 ACH	8.97	0.0511	2.06	41.38	491.53	0.825	0.580
PMA 6 ACH	9.93	0.105	2.20	21.85	595.6	1.00	0.744
NMD 12 ACH	6.23	0.311	2.29	8.36	377.79	1.269	1.145
SCD 12 ACH	2.64	0.0283	1.64	58.88	124.12	0.417	0.356
PMA 12 ACH	4.96	0.105	2.20	21.85	297.8	1.00	0.744

The above comparison should not be interpreted as showing that W alone controls short-term HCW exposure. The two 6 ACH cases illustrate this directly: their normalized spreads are nearly identical, $W = 2.09$ for NMD and 2.06 for SCD, yet their exposure rates differ substantially, $S_H = 0.805$ and 0.580 (Figure 14b). In particular, W measures only the width of the cumulative RTD and does not uniquely describe its early-time shape or the spatial transport of tracer near the HCW. The early-breakthrough ratio t_{10}/τ_{theor} , the complete cumulative-RTD curve, and the airflow topology must also be considered. The four cases nevertheless show a physically consistent pattern: the configuration with the clearest and most rapid patient-to-exhaust pathway has both a narrow cumulative removal response and a low rate of exposure accumulation near the HCW.

The PMA values are also shown in Figures 14b–d. In each panel the PMA point lies within the scatter of the four CFD cases about the fitted line, indicating that the perfectly mixed limit is consistent with these trends.

Figure 14c compares S_H with the normalized breakthrough time, $t_{10}/\tau_{\text{theor}}$. Within the four cases, later exhaust breakthrough is associated with a greater normalized HCW exposure rate, giving a descriptive $R^2 = 0.939$. The SCD 12 ACH case has both the earliest normalized breakthrough and the lowest S_H , whereas the NMD 12 ACH case has the latest normalized breakthrough and the highest S_H . Figure 14d shows the corresponding inverse relationship between S_H and the Morrill index, with $R^2 = 0.921$. This inverse trend should be interpreted carefully because $MI = t_{90}/t_{10}$; the large Morrill index of the SCD 12 ACH case is produced primarily by its very small t_{10} , not by greater late-time retention. Note that the comparisons in panels (c) and (d) are not independent because both metrics contain t_{10} .

Overall Figure 14 suggests complementary connections between exposure and cumulative-RTD behavior. Long-term HCW exposure is associated primarily with the overall tracer-retention timescale represented by τ_{CFD} . Short-term exposure reflects the early shape and breadth of the cumulative RTD, represented here by $t_{10}/\tau_{\text{theor}}$, $(t_{90} - t_{10})/\tau_{\text{theor}}$, and $MI = t_{90}/t_{10}$. These relationships provide a physical framework for interpreting the four simulations, but additional room configurations, ventilation rates, and source locations would be required to determine whether the observed trends constitute general quantitative correlations.

Because only four combinations of room configuration and ACH were simulated, the fitted lines in Figure 14 should not be interpreted as statistically established correlations or as predictive relationships applicable to other isolation rooms. Instead, they summarize how the exposure and RTD measures vary together within the present set of cases and help identify the transport mechanisms responsible for the observed exposure differences.

4 Limitations and Strengths

The results above have limitations. The transport equation in (10) is applicable to a passive tracer or potentially to aerosol particles smaller than 20 μm in diameter [26]. A full assessment of the HCW exposure should also consider aerosol particles of larger diameters, requiring Lagrangian particle tracking based on the conservation of momentum equation for individual particles [1, 26]. Processes such as evaporation, deposition, coagulation [33] and particle drift (due to gravity, thermal force by thermophoresis, particle fluctuation due to turbulence and particle acceleration) [34] should be important as well. For example, particle drift could cause some aerosol to escape the influence of short-circuiting. Future research should assess the influence of the above factors on HCW exposure and overall RTD indexes numerically and experimentally. Finally, simulations with additional patient postures and HCW locations should be made and different ACH values should be explored to strengthen the results obtained, although the ACH values considered in the current study (ACH=6 and 12) do cover the typical range in isolation rooms [5].

Notwithstanding these limitations, RTD analysis is inexpensive relative to the alternatives it complements. Numerically, a passive-scalar calculation requires one steady flow solution followed by a single unsteady scalar transport, at a small fraction of the cost of Lagrangian tracking across a range of particle sizes and release conditions. Empirically, the same indices can be obtained from a physical tracer-gas experiment (e.g. using SF_6) rather than from instrumented aerosol measurements: a pulse released at the patient's bed and sampled continuously at the exhaust yields a concentration history directly analogous to the RTD curves computed here, from which t_{10} , t_{90} , τ , W and MI follow. RTD analysis is therefore well suited as a screening tool for comparing candidate exhaust placements during design and for verifying that an as-built room performs as intended at commissioning, with higher-

fidelity multiphase simulation or physical aerosol testing both with room occupants reserved for the configurations that warrant it.

5 Conclusions

CFD simulations of patient-emitted tracer transport in a mock hospital isolation room were conducted for normal and short-circuiting exhaust configurations at 6 and 12 ACH. RTD analysis was used for the first time to characterize how exhaust placement and ventilation rate influence early breakthrough, late-time retention, room-scale clearance, and HCW exposure. The principal findings are as follows:

- (1) At 6 ACH, the NMD and SCD cumulative RTDs were relatively similar and remained close to the perfectly mixed response. Their normalized spreads were $W = 2.09$ and 2.06 , respectively, against 2.20 for the PMA, and their normalized breakthrough times, 0.136 and 0.0511 , bracketed the PMA value of 0.105 . The CFD mean residence times, 9.70 and 8.97 min, were likewise close to the theoretical 9.93 min. Changing exhaust placement at the lower ventilation rate therefore produced only a modest change in the overall tracer-removal response, although long-term HCW exposure still fell by approximately 20%, from 1.04 to 0.83 times the PMA value.
- (2) At 12 ACH, exhaust placement had a much greater influence. The SCD configuration established a direct patient-to-exhaust pathway, reducing the normalized breakthrough time from $t_{10}/\tau_{theor} = 0.311$ for NMD to 0.0283 for SCD. The normalized spread decreased from 2.29 to 1.64 , and τ_{CFD} decreased from 6.23 to 2.64 min. The two configurations fell on opposite sides of the completely mixed Morrill index of 21.85 , with $MI = 58.88$ for SCD and 8.36 for NMD. The large SCD value was caused primarily by its very small t_{10} rather than by a more persistent late-time tail, so the Morrill index must be interpreted together with t_{10} , t_{90} and the normalized spread.

- (3) Increasing ACH did not, by itself, guarantee a comparable reduction in short-term HCW exposure. The lowest normalized exposure rate, $S_H = 0.356$, occurred in the SCD 12 ACH case, in which the higher ventilation rate was combined with a direct patient-to-exhaust pathway. The NMD 12 ACH case instead produced the highest rate of the four, $S_H = 1.145$, exceeding both its own 6 ACH counterpart (0.805) and the perfectly mixed value of 0.744. Doubling the ventilation rate in the normal configuration therefore accumulated early exposure faster than a well-mixed room would, demonstrating that the effect of ACH depends strongly on airflow topology.
- (4) Increasing ACH reduced long-term (asymptotic) HCW exposure in both room design configurations, although the benefit was substantially greater for SCD: approximately 39% for NMD versus 75% for SCD. At 12 ACH, changing from NMD to SCD reduced the asymptotic exposure from 377.79 to 124.12 s, approximately 67%. Normalized by τ_{theor} , the four cases span 1.04 and 0.83 at 6 ACH to 1.27 and 0.42 at 12 ACH, so exhaust placement moved the exposure to either side of the perfectly mixed prediction at the same ventilation rate.
- (5) Across the four cases, long-term HCW exposure increased with τ_{CFD} , while the normalized short-term exposure rate varied systematically with the normalized cumulative-RTD spread, normalized breakthrough time, and Morrill index. With only four configurations these relationships are descriptive rather than predictive, but they indicate which RTD metric is diagnostic of which exposure timescale.

This work establishes RTD analysis as a quantitative framework linking airflow topology to the transport and clearance of patient-discharged air in a hospital isolation room. RTD analysis translates the complex flow dynamics into physically interpretable time scales for evaluating transport and room-scale removal of patient-discharged air and, secondarily, HCW exposure over short and long durations. Specifically, RTD analysis was shown to provide quantification of the “path principle” introduced by

Memarzadeh and Xu [5] (that a direct patient-to-exhaust pathway or short-circuiting design controls exposure) via measurable, design-usable metrics derived from the RTD curve and its cumulative form.

Results obtained suggest that instead of treating ACH as a proxy for performance, hospital isolation room designers should compute (or measure) the cumulative RTD and report t_{10} , t_{90} , τ_{theor} , and RTD-based τ , favoring layouts with low t_{10}/τ_{theor} and W (for robust short-circuiting) and low τ (for fast overall clearance). These RTD metrics help distinguish rapid patient-to-exhaust transport from slower removal governed by room-scale mixing, recirculation, and turbulent dispersion. RTD analysis can be based on CFD or physical tracer tests, providing a portable, ACH-agnostic basis to compare exhaust placements and room configurations in practice.

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7 Author Contributions

Anthony J. Pérez and Juan Peñaloza-Gutiérrez performed the computational simulations. Tauhidur Rahman contributed to the conceptualization of the study. Andrés E. Tejada-Martínez acquired the funding, conceptualized the study, analyzed and interpreted the results, and prepared the original manuscript draft.

8 Competing Interests

The authors have no relevant financial or non-financial interests to disclose.

9 Ethics Approval

Not applicable. This study involved only computational simulations of a mock hospital isolation room and did not involve human participants, identifiable human data, human biological materials, or animals.

10 Data Availability

The simulations and data supporting the findings of this study are available from the corresponding author upon reasonable request.

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