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Quantifying the reduction of airborne infectious viral load using a ventilated patient hood

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SUMMARY

Background: Healthcare workers treating SARS-CoV-2 patients are at risk of infection by respiratory exposure to patient-emitted, virus-laden aerosols. Source control devices such as ventilated patient isolation hoods have been shown to limit the dissemination of non-infectious airborne particles in laboratory tests, but data on their performance in mitigating the airborne transmission risk of infectious viruses are lacking.
Aim: We used an infectious airborne virus to quantify the ability of a ventilated hood to reduce infectious virus exposure in indoor environments.
Methods: We nebulized 10^9 plaque forming units (pfu) of bacteriophage PhiX174 virus into a ~30-m^3 room when the hood was active or inactive. The airborne concentration of infectious virus was measured by BioSpot-VIVAS and settle plates using plaque assay quantification on the bacterial host *Escherichia coli C*. The airborne particle number concentration (PNC) was also monitored continuously using an optical particle sizer.
Findings: The median airborne viral concentration in the room reached 1.41×10^5 pfu/m^3 with the hood inactive. When active, the hood reduced infectious virus concentration in air samples by 374-fold. The deposition of infectious virus on the surface of settle plates

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was reduced by 87-fold. This was associated with a 109-fold reduction in total airborne particle number escape rate.

Conclusion: A personal ventilation hood significantly reduced airborne particle escape, considerably lowering infectious virus contamination in an indoor environment. Our findings support the further development of source control devices to mitigate nosocomial infection risk among healthcare workers exposed to airborne viruses in clinical settings.

Introduction

The treatment of patients with coronavirus disease 2019 (COVID-19) has led to considerable risk of infection in healthcare workers (HCWs) with associated morbidity and mortality [1,2]. Acute respiratory syndrome coronavirus 2 (SARS-CoV-2) can be spread by fomites, droplets and aerosol particles [2,3]. Hazard management strategies favour engineering methods (e.g., control of airflows) to isolate people from the source of risk over personal protective equipment (PPE) [4]. Hospital environmental engineering controls primarily rely upon isolation rooms with specialized ventilation systems to limit the spread of airborne viruses outside of designated areas. However, these facilities are a scarce resource, may not fully contain SARS-CoV-2, and importantly, may not entirely protect HCWs working within the room [5]. In lieu of this, much focus has been placed upon correct PPE use by HCWs (e.g., N95 mask and barrier gowns) to reduce spread of SARS-CoV-2 to HCWs and other patients [3]. However, despite these measures, HCWs have remained at high risk of nosocomial illness following SARS-CoV-2 exposure in healthcare settings [6].

To improve protection for HCWs, engineering control devices such as portable air cleaners are increasingly being deployed in concert with existing infection control measures to enhance the clearance of contaminated indoor air [7,8]. Directly controlling the emission source of infectious aerosols would be ideal to reduce the risk of airborne transmission. However, there are limited methods to contain the respiratory aerosols emitted by infectious patients. For example, placing an N95 mask on the patient often cannot be done without compromising their care. Previously, we developed a patient isolation hood (McMonty hood) [9] and tested its utility in containing the emission of physical aerosols by at least 98% in a laboratory environment [10]. Its efficacy in reducing the airborne viral load in a room can be inferred from these tests, but the McMonty hood has not be validated experimentally using an infectious agent.

The bacteriophage PhiX174 (family *Microviridae*) is a small (25 nm diameter, approx. ¼ SARS-CoV-2's size) [11], non-enveloped, bacteriophage with a linear ssDNA genome that is harmless to humans and is routinely used as a surrogate pathogen for the study of airborne viral transmission [12–14]. Landry *et al.* [15] recently quantified viable airborne PhiX174 virus propagated from a positive airway pressure circuit leak, and nebulized viral aerosols were successfully contained by a makeshift plastic hood cover and a commercial HEPA filter with a fan. We tested how effectively the McMonty patient isolation hood could contain an airborne virus emission source by nebulizing PhiX174 to quantify the ability of the hood to limit the level of infectious aerosol exposure for HCWs in clinical settings [16].

Methods

Ethics approval for this laboratory-based study was deemed not required by Monash University's School of Biological Sciences Ethics Manager.

Bacteriophage propagation and quantification

Bacteriophage PhiX174 was propagated on its bacterial host *Escherichia coli C* (ATCC13706) grown in lysogeny broth (LB). Viral product was purified from lysate using the Phage-on-Tap protocol [17] and re-suspended in phosphate-buffered saline (PBS, Omnipur®, Merck, Gibbstown, NJ, USA). The viable concentration of PhiX174 virus stocks was quantitated by plaque assay using the soft agar overlay method [17]. Viable counts of PhiX174 were expressed as plaque forming units per millilitre of suspension (pfu/mL).

Patient isolation hood

The McMonty isolation hood consists of a mobile steel frame, a plastic canopy, and an extraction fan equipped with a standard high efficiency particulate air (HEPA) H13 filter (rated to 99.95% clearance of 0.3-μm particles). The plastic barrier opens out to form a hood with 1.3 m^3 internal volume, enclosing the patient's torso from the waist up (non-airtight seal). The extraction fan (Westaflex, Melbourne, Australia) is mounted behind and above the patient's head. It draws the air from around the patient, passing it through a HEPA filter (Techtronic Industries, Hong Kong, China) before recirculating clean air to the surroundings (clean air delivery rate: CADR, at 40L/s (144 m^3/h); equivalent to 110 air changes per hour (ACH) within the hood) [10].

Airborne virus containment using the McMonty hood in an indoor space

The McMonty isolation hood was tested in an approx. 4 × 3.25 × 2.3 m (29.9 m^3) room that contained a single bed with the hood positioned over a simulated patient (Figure 1a). PhiX174 bacteriophage was aerosolized into the sealed room to mimic shedding of airborne virus. The virus emission source was simulated using a nebulizer (Pari-PEP®, PARI Respiratory Equipment, VA, USA) placed where the head of the patient would rest (20 cm above surface of bed; see Figure 1b, location N) with the outlet facing upwards. The Pari-PEP device produced aerosol particles with unimodal polydisperse size distribution with the mass median diameter of 3.42 ± 0.15 μm [18]. The room ventilation ports were covered to avoid viral egress (i.e., no active heating, ventilation, and air-conditioning

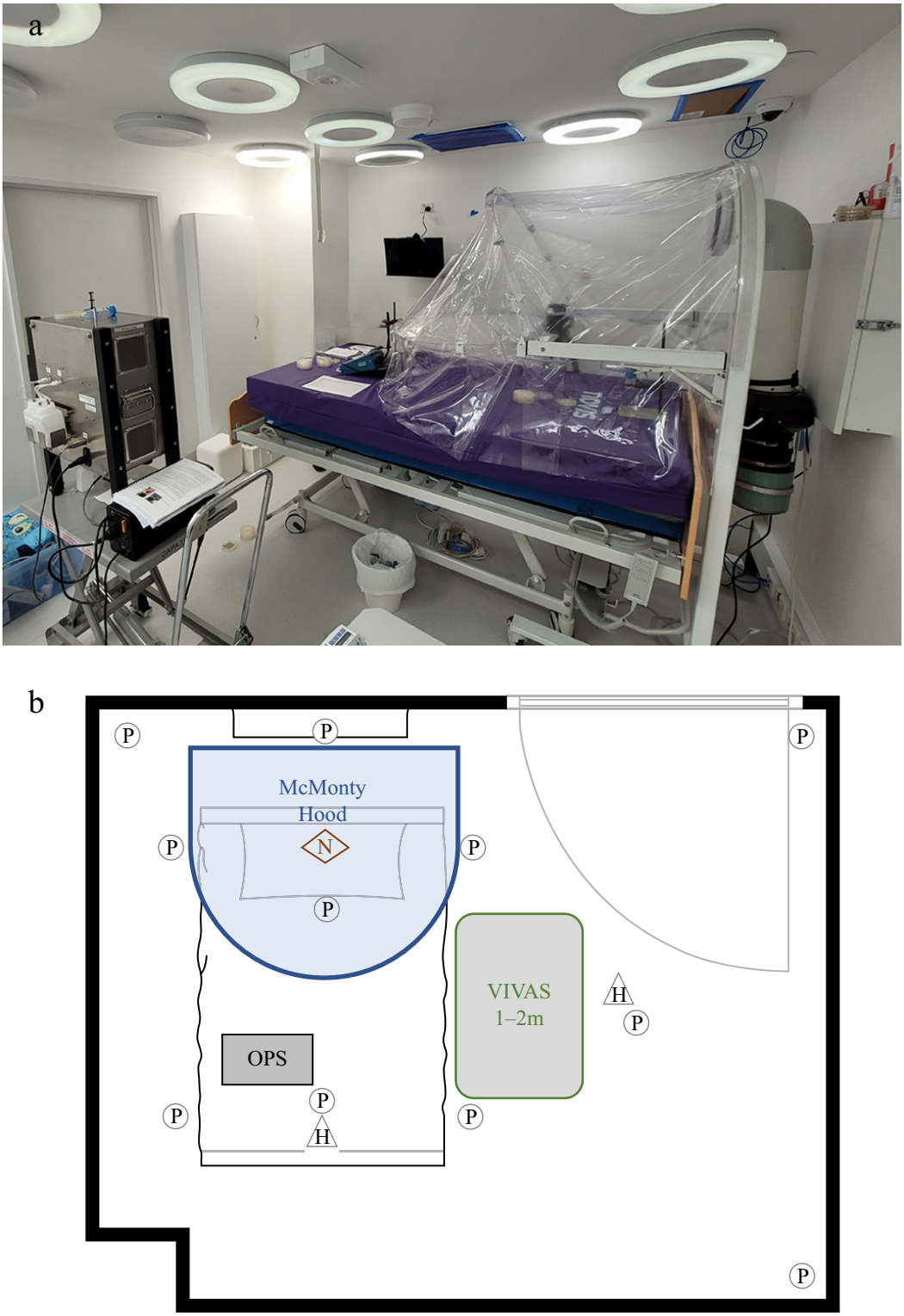

Figure 1. Indoor test room configuration for the McMonty personal isolation hood. (a) Photograph of experiment room with the McMonty hood deployed in active mode. The BioSpot-VIVAS and settle plates are positioned for testing. (b) Schematic of indoor test room (approx. dimensions 4.0 × 3.25 × 2.3 m, volume = 29.9 m^3). Eleven settle plates (circles, P) were arranged on room surfaces; two hanging plates (triangles, H) were suspended at head height (175 cm) with exposed faces directed towards the bed. The nebulizer (diamond, N) was positioned at the head of the bed, with the exit point facing vertically upwards. Air sampling was performed using a BioSpot-VIVAS positioned at the bedside 1–2 m from the nebulizer. Particle concentration was assessed by optical particle sizer (OPS) positioned in the centre of the bed, outside the hood. Virus and aerosol measurements were taken when the nebulizer was actively contained by the hood (i.e., hood physically isolated nebulizer from instruments), compared with when no containment was used (i.e., instruments were exposed to nebulizer).

(HVAC)) to ensure changes in detected airborne virus resulted from the activity of the McMonty hood. Sampling devices were arranged around the room to detect the spread of airborne particles laden with PhiX174 (Figure 1b). We continuously monitored room temperature and relative humidity during all experiments, at 21.6–27.0 °C (mean 24.7 °C) and 46–67% (mean 53.1%), respectively.

Aerosol generation experiments were conducted four times each day over three independent days. We nebulized 10^9 pfu (10 mL at 10^8 pfu/mL) of bacteriophage over a 40-min generation period. In all experiments the nebulizer was positioned on the bed to simulate a patient receiving care. Samples were collected in the room for two test conditions: (1) In the 'no containment' condition, the isolation hood was not deployed to enclose the bed and nebulizer, and the extractor fan/HEPA filter was inactive during the generation period. These conditions simulate a patient in an open room. Bacteriophage samples collected in the room during this condition demonstrate the baseline airborne viral contamination generated by the nebulizer without mitigation measures. (2) In the 'McMonty active' condition, the isolation hood was deployed to enclose the nebulizer (a non-airtight plastic skirt covering the top half of the bed) with the extractor fan/HEPA filter turned on during the generation period. These conditions simulated a patient with an isolation hood placed over them as a mitigation measure.

We aimed to compare the detection of bacteriophage in the room between the active and no containment conditions to calculate the fold-change in airborne viral contamination resulting from the use of the hood. After each condition the room air was purged of residual airborne particles by running a portable air purifier (IQAir HealthPro 250, Goldach, Switzerland) for 30 min at a CADR of 470 m^3/h (15.7 ACH) before beginning the next condition (Supplementary Figure S1).

Detection of airborne infectious PhiX174

We employed the passive settle plate detection method for nebulized PhiX174 established previously by Landry *et al.* [15] Thirteen settle plates containing agarose pre-inoculated with *E. coli C* bacterial host were positioned in the room and exposed during each aerosol generation period (Figure 1b). In each set, 2/13 settle plates were suspended from the ceiling at approximate head height (175 cm from the ground) with exposed surface facing the head of the bed. Settle plates were incubated at 37 °C overnight and plaque counts enumerated per plate to detect the deposition of airborne particles laden with infectious bacteriophage virus.

In parallel, the BioSpot-VIVAS 300-P (VIVAS; Aerosol Devices, Fort Collins, CO, USA) was used to actively sample particles from air in the room at 8 lpm. The VIVAS was positioned adjacent to the hospital bed and its air sample intake was located 1–2 m away from the nebulizer (Figure 1b), simulating the position of a HCW at the bedside. Each air sample was collected as condensate into a Petri dish (35 mm diameter) containing 3 mL of sterile PBS, which was stored at 4 °C until analysis. The infectious titre of PhiX174 in VIVAS sample fluid was determined by plaque assay as described above. VIVAS detection of airborne virus was expressed as pfu per cubic metre of air collected during the 40-min nebulization period (pfu/m^3). The VIVAS inlet and sampling lines were decontaminated (Supplementary Figure S2) by flushing with 70% ethanol then distilled water prior to each subsequent sample.

Aerosol monitoring instrumentation

Airborne particle number distribution and concentration were monitored during each experiment using an optical particle sizer (OPS, TSI Model 3300), which allows detection and size classification of aerosol particles within a 0.3- to 10-μm-diameter range. McMonty hood performance was assessed using total particle number concentration (PNC). The instrument was placed outside of the hood at the foot of the patient bed with sampling inlet 0.2 m above the bed (Figure 1b) and logged measurements at 10-s average intervals. The OPS was present for two of three independent experimental days (Supplementary material), and one room sample with the McMonty hood active was excluded from analysis due to a protocol deviation (Supplementary Figure S3).

Data analysis

Infectious phage samples from each experiment were categorized based on their relative exposure to the nebulizer with the McMonty hood active or inactive. Data from all samples were treated as experimental replicates and pooled according to containment condition. Settle plates counts exceeding the limit of detection (too many to count, TMTC) were included as 300 pfu. The untransformed pfu counts (settle plate) or pfu/m^3 (VIVAS) were compared between each containment condition by the Wilcoxon rank sum test (R version 4.1.1, The R Foundation for Statistical Computing, Vienna; package 'rstatix'). *P*-values <0.05 were considered statistically significant. Wilcoxon effect size (r) was calculated as Z score/$\sqrt{}$(sample size). For both settle plate and VIVAS data, we compared the fold-change in median values for McMonty hood active versus inactive datasets.

We defined a theoretical model to describe the PNC inside the room throughout each experiment (Supplementary material). The model explains rate of change in PNC over time (per minute) through a combination of the number of airborne particles: (1) escaping the hood, and (2) in the room lost through deposition. Experimental PNC data measured by the OPS was fitted to this model to calculate the rate of particles escaping into the room when the McMonty hood was active versus inactive. These values were used to calculate the effective filtration efficiency, expressed as the percentage reduction of the particle escape rate.

Results

Patient isolation using the McMonty hood reduces concentration of airborne infectious virus

Figure 2 shows the airborne concentration of infectious PhiX174 in the room using an active McMonty hood compared with an inactive control condition (no containment). With active containment of the emission source, the median concentration of viable virus in air samples was 3.77×10^2 pfu/m^3 ($N = 6$, interquartile range (IQR) = 262–1277) compared with 1.41×10^5 pfu/m^3 ($N = 7$, IQR = 33,750–182,500) without containment. This equates to a significant 374-fold reduction in airborne infectious virus contamination ($W = 0$, $P < 0.005$, $r = 0.83$).

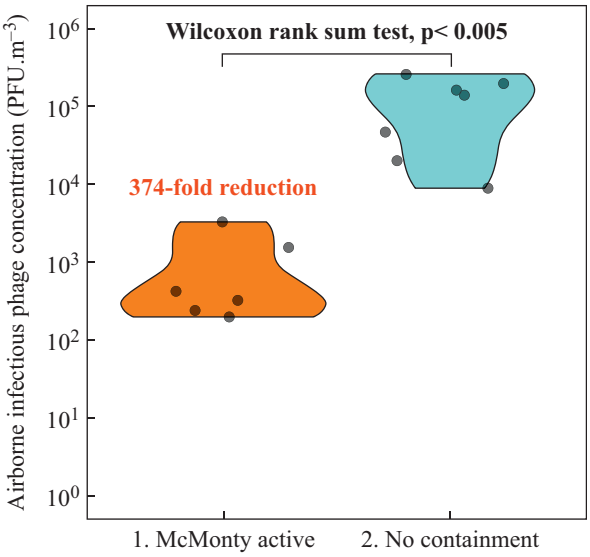

Figure 2. Violin-plot of viable airborne phage concentration (pfu/m^3) detected by BioSpot-VIVAS (y-axis) in each containment condition (x-axis). Distribution of individual measurements (black points) of bacteriophage plaque forming units per m^3 of air (pfu/m^3) when the McMonty hood was inactive during the sampling period (right column) compared with measurements taken when active hood source containment was used (left column). Annotated 374-fold reduction in median airborne virus concentration when McMonty hood was active. Groups were compared (untransformed values) by Wilcoxon rank sum test.

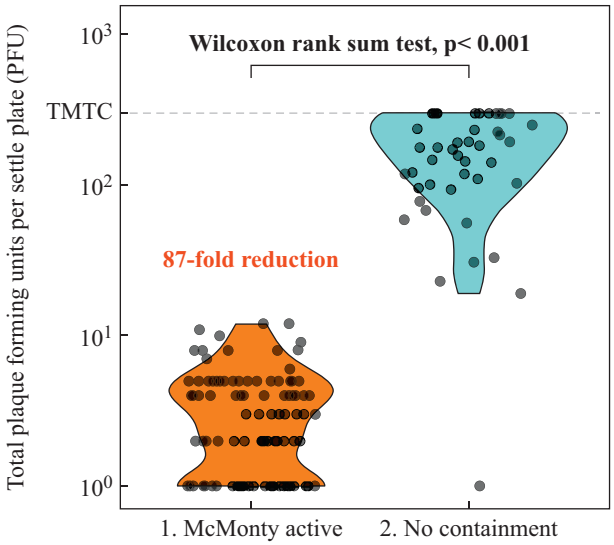

Figure 3. Violin-plot of total viable phage count (pfu) detected on settle plates (y-axis) in each containment condition (x-axis). Distribution of individual plaque forming unit (pfu) counts (black points) for each settle plate when no containment (inactive hood) was used during the sampling period (right column) compared with counts reached when active McMonty hood containment was used (left column). Counts transformed by +1 pfu to visualize 0 pfu plates on the $\log_{10}$ scale (on graph only). Plates with plaque counts exceeding the saturation point on each plate were given the value of 300 for statistical tests (threshold labelled as too many to count; TMTC). Annotated 87-fold reduction in median settle plate count when McMonty hood was active. Groups were compared (untransformed values) by Wilcoxon rank sum test, $P < 0.05$ indicates statistically significant difference. Outliers were excluded from violin-plot visualizations but are visible in the scatterplot.

Infectious virus counts from airborne particles which deposited on to the surface of settle plates

Infectious virus counts on settle plates were reduced with active source containment (<11 pfu), and 33/108 plates did not detect any viable phage (Supplementary Figure S4). Active isolation hood containment significantly reduced the median settle plate count (median = 2, $N = 108$ IQR = 0–4) by 87-fold ($W = 91.5$, $P<0.001$, $r = 0.775$) compared with plates without source containment (median = 174, $N = 48$, IQR = 94.5–300) (Figure 3). Phage counts on plates suspended from the ceiling ($N = 6$) did not differ from the trends observed in the overall settle plate dataset.

Effect of the active McMonty hood upon containment of airborne particles

Figure 4 shows the change in airborne PNCs over time when the hood was in use compared with the no containment condition. Datasets were fitted to a model (Supplementary material) to quantify the rate of aerosol particles escaping into the test room in each containment condition, with good reproducibility and high correlation of measurements with the model (adjusted r^2 values: active = 0.86, inactive = 0.96).

A two-orders of magnitude decrease in PNC were observed within the room when the McMonty hood was active (Figure 4). This reduction was also reflected in the modelled escape rates (Table I) with a 109 ± 5 fold reduction in the rate of viral aerosol escape into the room when the hood is active. Effective filtration efficiency of the hood (Supplementary material) indicated that the active McMonty hood removed 99.1 ± 0.1% of viral aerosol released.

Discussion

In an indoor test environment, we nebulized a high titre bacteriophage (PhiX174) suspension, comparing the dissemination of infectious phage and airborne PNCs when a personal isolation hood was active/inactive. We detected a 109-fold reduction in the escape of total airborne particles into the room when the hood was in use. Importantly, this was correlated with a 374-fold decrease in the airborne concentration of infectious virus, and an 87-fold reduction of infectious contamination for surfaces in the room.

Despite enhanced infection prevention (including PPE) hospital protocols during the COVID-19 pandemic, nosocomial infections among HCWs continued to strain hospital systems worldwide. To augment existing infection control strategies, researchers have pursued aerosol containment at the point of emission with personal isolation units [19], including ventilated headboards [20], and similar stationary devices [21] attached to building ventilation units. Nishimura *et al.* atomized influenza virus inside their Barrihood isolation fan/filter unit, finding that none escaped [22]. Using a PhiX174 model similar to our study, Landry *et al.* showed that a makeshift isolation hood greatly augments the protection conferred by standard

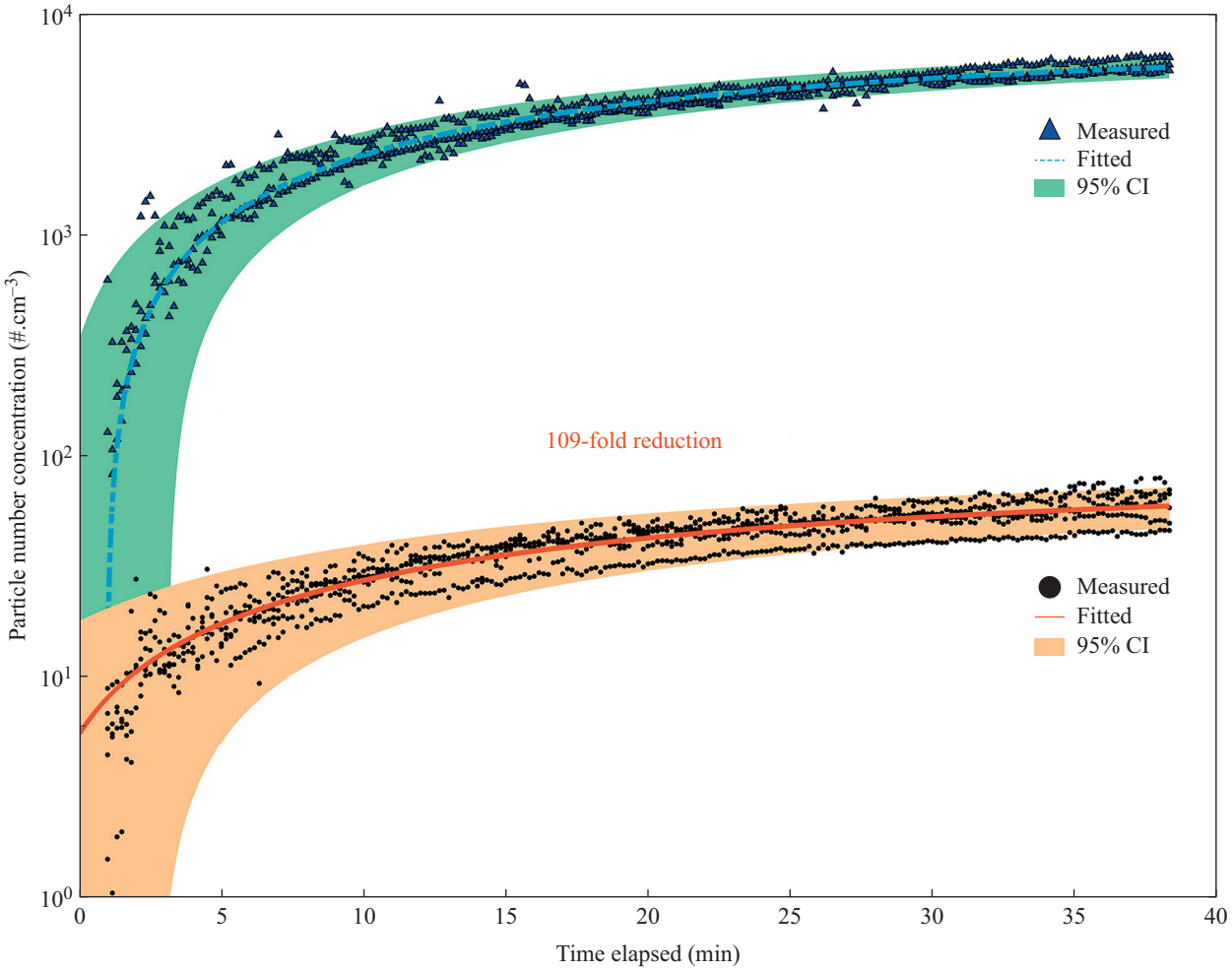

Figure 4. Airborne particle number concentration (PNC) measurements of the test room during bacteriophage nebulization. Data from the optical particle sizer (OPS) was grouped based on containment type and fitted to the theoretical model (Supplementary material) with 95% confidence interval (CI, shaded area). Data measurements are shown for when the McMonty hood was active (circles) compared with inactive (triangles), labelled with the fold-reduction number from the particle number escape rate calculated in the fitted model.

Table I
Summary of physical and infectious aerosol results from optical particle sizer and VIVAS. Fitted values of viral aerosol escape rates (mean ± standard deviations) with the McMonty hood active or inactive are presented, with the calculated fold reduction and effective filtration efficiency. Median virus concentration (± median absolute deviation) measurements are provided for VIVAS virological data; corresponding fold reduction and percentage reductions are calculated from the median values

Physical aerosol analysis \ 1	McMonty active escape rate (log_{10} #/min)	Inactive (no containment) escape rate (log_{10} #/min)	Fold reduction	Effective filtration efficiency (%)
	7.99 ± 6.60	10.02 ± 8.48	109 ± 5	99.1 ± 0.1
Virological aerosol analysis \ 1	**McMonty active viral conc. (median) (log_{10} pfu/m^3)**	**Inactive viral conc. (median) (log_{10} pfu/m^3)**	**Fold reduction**	**Percentage reduction (%)**
	2.58 ± 2.36	5.15 ± 5.14	374	99.7

hospital PPE by limiting skin exposure to viral aerosols [23]. Despite the experimental efficacy of these isolation hood concepts, such devices have not been in widespread clinical use during the COVID-19 pandemic.

Concessions to human comfort and ergonomic requirements must be addressed before the widespread clinical adoption of isolation hoods. The McMonty isolation hood was designed to balance the patient's tolerance for high rates of airflow and

noise [24] against the device's effectiveness, while cognisant that lower air flow rates reduce the efficacy of clearing infectious respiratory agents. We demonstrated that the McMonty hood could significantly reduce the emission of physical aerosols into the surrounding room while maintaining lower (<0.5 m/s), more tolerable airflow rates near the patient's head [24]. Importantly, we correlated the reduction of physical particle counts by ventilation with a reduction of viable virus concentration in the room air and on room surfaces, providing a more relevant metric to assess the hood's potential to prevent HCW exposure to infectious virus. A consequence of the hood's airflow is the intake of unfiltered air from the surrounding room, which may carry additional airborne pathogens that pose a threat to the patient. However, the high rate of air exchange for the hood's overall air volume (110 ACH) ensures that aerosols generated by the patient or drawn into the hood from the surrounding room are promptly ventilated and do not accumulate near the patient.

Our study design has inherent limitations. Bacteriophage PhiX174 was selected as a surrogate viral agent, where we ideally would have tested the hood directly against SARS-CoV-2 in a hospital setting. However, such a clinical approach would: (1) have exposed research staff to patients with COVID-19; (2) be uncontrolled, i.e., there would be considerable variability in aerosol emission between different patients; and (3) be uncertain to produce sufficient airborne virus levels to accurately quantify the performance of the active hood, as the level of viral escape from a human patient in a hood would likely be below the limit of detection for our instruments. Most relevant nosocomial viruses possess an envelope, e.g., coronaviruses and influenza viruses, and evidence suggests that the viral envelope is one of a complex range of factors which influence environmental stability of infectious aerosols, including the chemical microenvironment of the droplet and its interactions with viral surface structures [25]. With these factors in mind, our compromise was to select PhiX174 as a non-enveloped surrogate virus that had been established with reliable performance in our test environment, and had previously shown similar or enhanced characteristics of surface and aerosol stability, respectively, to enveloped bacteriophages such as Phi6 in comparable conditions [7,13,15,25].

We designed a stringent test scenario to enable reproducible and detectable measurements of the performance of the hood. The bacteriophage nebulization protocol we employed generated a median airborne viral concentration (1.41×10^5 pfu/m^3 without source containment) that is approximately two orders of magnitude greater than the level of exhaled infectious SARS-CoV-2 generated by patients (8.9×10^2 $TCID_{50}$/m^3) in recent reports [26]. Our amplified viral challenge resulted in the detection of approx. 10^3 pfu/m^3 airborne phage escaping the active hood, enabling the calculation of the hood's effectiveness by fold-reduction. We performed our tests in the absence of standard HVAC air circulation to ensure we were measuring differences resulting from the activity of the hood alone. While this environment does not reflect the conditions of most state-of-the-art environments, it is more analogous to 'worst-case' scenarios for infection control, e.g., in facilities with suboptimal ventilation. We anticipate that a similar ratio of infectious airborne virus measured with the hood inactive/active would be achieved in standard hospital rooms, as active HVAC would uniformly extract additional virus-laden air regardless of hood operation, and the generation of lower airborne virus concentrations from the same volume of nebulized material (Supplementary Figure S2) should not impact the effectiveness of the hood's extractor fan.

We quantified the exposure of airborne infectious virus in the room using two techniques to address different risk factors in an indoor clinical setting. The respiratory exposure risk of medical staff in virus-infected patient rooms can be approximated by the bedside placement of the VIVAS which samples air at 8 lpm, similar to human minute ventilation rates [27]. The deposition of infectious viral aerosols on to the surface of settle plates can similarly approximate the potential risk of fomite generation on contaminated room surfaces. The 40-min sampling period is probably insufficient to measure the total amount of infectious virus settling on surfaces and, unlike humans, the nebulizer does not produce larger droplets that favour surface deposition [28,29]. However, with a repeated measures design, we were able to assess the relative reduction in viral deposition between hood active and inactive conditions. Furthermore, the agreement between the settle place findings, air sampling and particle concentration measures provide us with confidence in the accuracy of the assessments. In our test system, the McMonty hood substantially reduced viral exposure by both airborne and surface metrics.

In conclusion, we have shown that a personal isolation hood can effectively reduce viral and aerosol escape by >99% in a simulated indoor healthcare setting. Along with our recent clinical study of ease-of-use [30], these findings support the further development of source control devices to limit HCW exposure to airborne virus. Future studies could elucidate the hood's risk mitigation potential using more relevant clinical parameters, with more realistic environmental airflows and airborne virus load. Undertaking a randomized, controlled clinical trial of the efficacy of isolation hoods (or of air cleaners) in preventing HCW SARS-CoV-2 infections is likely to be overwhelmingly challenging due to the large sample size required. Smaller-scale studies have shown the potential for complementary engineering methods to clean indoor air environments, e.g., using HEPA-filtered air cleaners in hospitals [7,8] to reduce the risk of airborne viral transmission. Our study using the McMonty hood supports the further consideration of such source control devices in risk mitigation strategies to prevent HCW nosocomial infections, helping to relieve the strain on healthcare systems.

Conflict of interest statement

The University of Melbourne and Western Health have patented the McMonty Hood, and authors F.M. and J.M. have received royalties for associated sales. The manufacturers of the McMonty Hood had no role in this study's design nor manuscript preparation. All other authors have no conflicts of interest to declare.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jhin.2023.04.009.

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