

Quantifying Environmental Mitigation of Aerosol Viral Load in a Controlled Chamber With Participants Diagnosed With Coronavirus Disease 2019

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Background. Several studies indicate that coronavirus disease 2019 (COVID-19) is primarily transmitted within indoor spaces. Therefore, environmental characterization of severe acute respiratory syndrome coronavirus 2 viral load with respect to human activity, building parameters, and environmental mitigation strategies is critical to combat disease transmission.

Methods. We recruited 11 participants diagnosed with COVID-19 to individually occupy a controlled chamber and conduct specified physical activities under a range of environmental conditions; we collected human and environmental samples over a period of 3 days for each participant.

Results. Here we show that increased viral load, measured by lower RNA cycle threshold (C_T) values, in nasal samples is associated with higher viral loads in environmental aerosols and on surfaces captured in both the near field (1.2 m) and far field (3.5 m). We also found that aerosol viral load in far field is correlated with the number of particles within the range of 1–2.5 μm . Furthermore, we found that increased ventilation and filtration significantly reduced aerosol and surface viral loads, while higher relative humidity resulted in lower aerosol and higher surface viral load, consistent with an increased rate of particle deposition at higher relative humidity. Data from near field aerosol trials with high expiratory activities suggest that respiratory particles of smaller sizes (0.3–1 μm) best characterize the variance of near field aerosol viral load.

Conclusions. Our findings indicate that building operation practices such as ventilation, filtration, and humidification substantially reduce the environmental aerosol viral load and therefore inhalation dose, and should be prioritized to improve building health and safety.

Keywords. SARS-CoV-2; aerosol; infectious disease; airborne; particles; ventilation; humidification; filtration.

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the causative agent of coronavirus disease 2019 (COVID-19), has resulted in 274 472 724 confirmed cases with more than 5 012 337 deaths globally, as of 3 November 2021 [1]. There is substantial evidence that inhalation of aerosol particles containing viable SARS-CoV-2 virions is the primary route of human-to-human transmission [2–9]. Modeling of the impact of nonpharmaceutical interventions on the probability of COVID-19 infection and mortality rate [10–14] suggests that indoor congregation is the primary driver for COVID-19 disease transmission [15]. Moreover, recent comprehensive reviews highlight the importance of airborne transmission pathway via fine aerosols [16–18]. Therefore, better understanding and quantification of the relationship of human

factors, design, and building operation practices on the abundance and dispersion of viral load in indoor spaces is necessary to combat disease transmission [19].

Breathing and talking are some of the human expiratory activities that have been studied to determine how these activities are associated with concentrations of viral pathogens [20, 21]. These studies have contributed valuable information about the viral load of size fractionated aerosols [5, 22]. In addition to human expiratory factors, indoor space design and engineering practices such as ventilation, filtration, and humidity control may influence the abundance and infectious fraction of the environmental viral load, and therefore reduce inhalation dose [22–28]. However, these indoor environmental interventions need to be studied independently through controlled experiments to quantify their impacts, while minimizing confounding variables, especially with regard to aerosols that may contain SARS-CoV-2.

In this research, we sought to better understand viral abundance and dispersion associated with differing degrees of expiratory activity, ventilation, filtration, and humidification through controlled experiments in a quasi-field setting. We measure viral RNA of SARS-CoV-2 using quantitative reverse-transcription

Received 4 November 2021; editorial decision 28 December 2021; published online 6 January 2022.

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Clinical Infectious Diseases® 2022;75(1):e174–84

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polymerase chain reaction (qRT-PCR) techniques as a proxy of viral load in humans and environmental aerosols and surfaces. We studied 11 human participants, who were diagnosed with COVID-19, in a controlled chamber measuring 4.3 m in length, 2.8 m in width, and 2.5 m in height (28.04 m³). Our research protocol comprised a 3-day study for each participant in which human activity and environmental factors (ventilation rate, in-room filtration, humidity control) were studied as independent variables.

MATERIALS AND METHODS

A rapid deployment modular unit (RDM) was used as an environmentally controlled chamber (Figure 1) for this human participant study during winter and spring 2021. The study population included 11 participants between the age of 18 and 24 (Supplementary Table 1). Two high-flow (200 L/min) AerosolSense air samplers (Thermo Fisher Scientific) were placed approximately 1.2 m and 3.5 m from the participants. At the end of each study period, samples from the air samplers (near, far), high-touch surfaces (phone, computer, chair), settling plates (near, far), and human specimens (shallow nasal) were collected and transported to a Biosafety Level 2 laboratory on the University of Oregon campus in Eugene, Oregon, for further molecular analysis.

Trials were conducted in 2 different setups over 3 days. Trials with a S1 suffix indicate Setup 1, where both air samplers were placed next to each other for short duration and higher expiratory tests (Figure 1A). During cough trials, participants were instructed to conduct 10 uncovered coughs into an area over the air samplers, particle counters (TSI AeroTrak 9306), and carbon dioxide (CO₂) (Onset HOBO MX1102A) sensors. During speak tests, participants were instructed to conduct continuous vocalization using a standardized Centers for Disease Control and Prevention-defined passage [29] (Supplementary Document, Appendix A) for 5 minutes with normal and higher amplitude at their discretion, respectively [30]. An S2 suffix indicates trials where air samplers were located at 1.2 m (near field) and 3.5 m (far field) of the participant's sitting position (Figure 1B). During S2 trials, participants conducted routine activities at a desk, including sitting and standing, sitting silently, and sitting and participating in an online conference meeting, or were invited to walk on a treadmill (physical activity day) (Figure 1B).

Institutional approvals, data availability, and methods related to RDM layout, participant recruitment, sample collection, molecular analysis, and statistical analysis are described in Supplementary Document, Appendix B.

RESULTS

Near and Far Field Aerosol Samples and Paired Human Specimens

To quantify the relationship between viral loads (RNA copies) in human nasal and aerosol samples, we paired the outcome of each aerosol sample collected with its corresponding shallow

nasal sample for both near and far AerosolSense samplers during trials when participants were sitting or standing for 1 hour at ~0 air changes per hour (ACH) under typical ambient conditions without environmental interventions. We defined routine trials according to the following conditions: (1) participants conducted typical office activity while sitting or standing for 1 hour; (2) ambient environmental conditions were maintained using only electric resistance heaters without ventilation at ~0 ACH; and (3) participants could have spontaneously coughed because of their symptoms but were not instructed to conduct any expiratory activity during routine trials. Figure 2A shows the relationship between nasal viral load and near field and far field aerosol viral load for all routine trials. Note that negative samples are defined with a C_T value of 40.

The coefficients associated with significant regression models presented in Figure 2A indicate that an increase in viral load equivalent to -1 C_T in human nasal samples is associated with increased near field viral load of -0.326 C_T (R² = 0.2276, P = .001092) and increased far field viral load of -0.40 C_T (R² = 0.4026, P = 1.721e-06). The difference of means between the aerosol C_T value of near field and far field aerosol samples was 1.058 C_T, indicating lower viral load for far field samples; however, the paired *t* test differentiating near field and far field samples was not significant (P = .05955) (Figure 2B; note that black solid horizontal line represents median in all box plots). Therefore, we also report the significant coefficient for all nasal and aerosol samples in routine trials, which indicates that an increase in viral load equivalent to -1 C_T in nasal samples is associated with an increase in room aerosol viral load of -0.362 C_T (R² = .3119, P = 1.675e-08; Supplementary Figure 1). Based upon qRT-PCR theory, a -1 C_T difference is approximately equivalent to double the viral load [31]; thus, a doubling of viral load in nasal samples corresponds to a ~35% increase in aerosol viral load for samples collected in the room. To our knowledge, this is the first reported relationship between environmental aerosol viral load and human viral load in a controlled environment (28 040 L³ room, ~0 ACH, 1-hour trials, single COVID-19-positive individual).

Furthermore, we found a statistically significant difference between the mean CO₂ concentration recorded at near field and far field, where CO₂ concentrations of near field were 80 parts per million (PPM) higher than in the far field (P = .0004009) (Figure 2C). Analysis of particles for routine trials indicates that there is a statistically significant difference between the number of particles collected in the range of 1–5 μm within the near field vs the far field, as summarized in Figure 2D (expanded in Supplementary Figure 2). As shown in Figure 2E, we identified a significant relationship between aerosol viral load and far field particle counts within the size bin 1–2.5 μm where increased number of particles within this size bin is associated with higher aerosol viral loads (R² = 0.1112, P = .04313). The relatively low reported R² is likely due to the reality that there

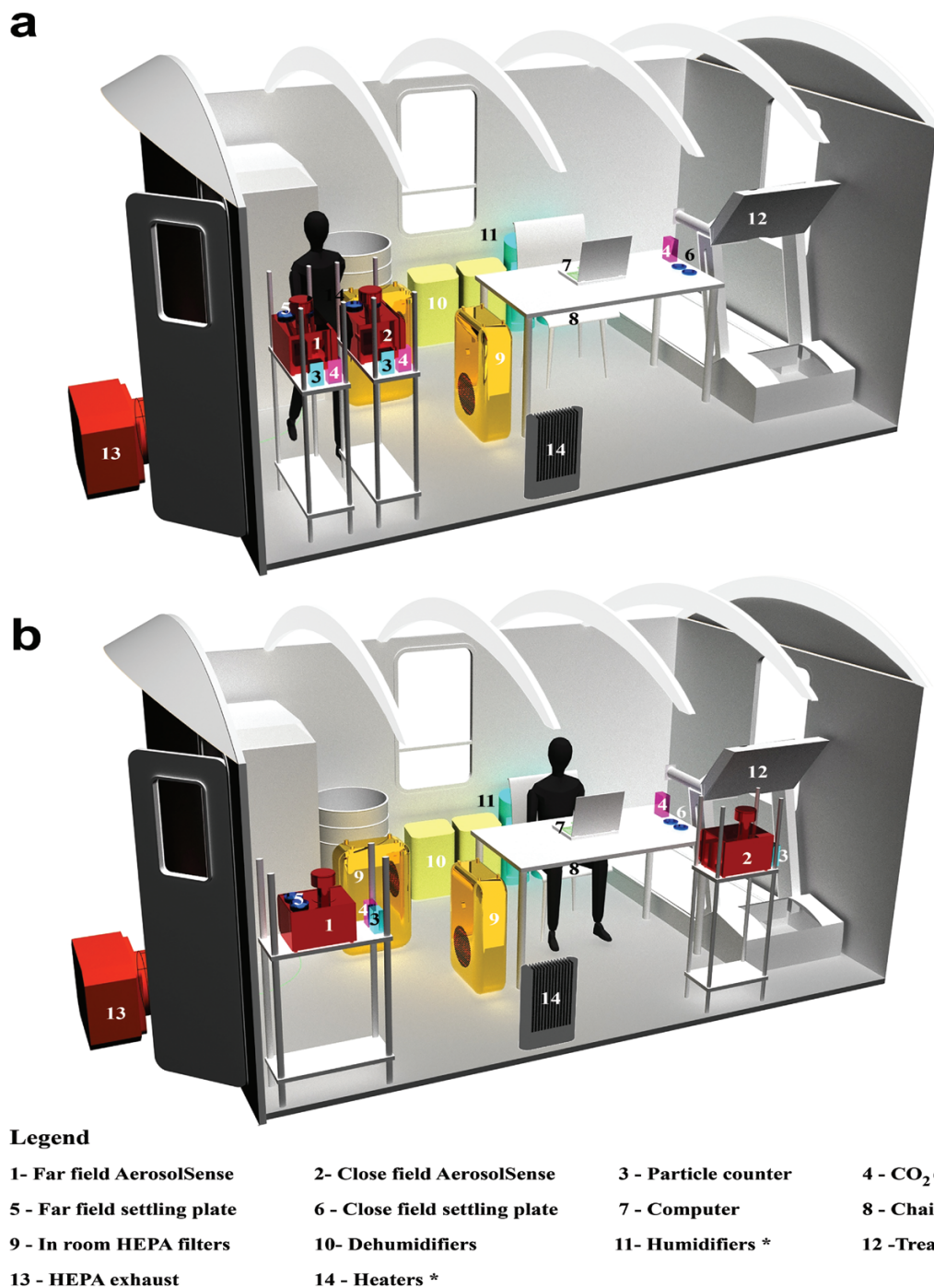


Figure 1. Rapid deployment modular unit (RDM) in higher expiratory trials (setup 1; A) and regular trials (setup 2; B). *Only 1 object of this kind is visible in this figure. Abbreviations: CO₂, carbon dioxide; HEPA, high-efficiency particulate air.

are many particles in the room that are not human-sourced bioaerosols, and therefore this regression should not be interpreted as an absolute prediction model. We also report a statistically significant positive correlation between the average far field CO₂ concentration and the number of particles of 0.3–3

μm in far field for routine trials (Supplementary Figure 3), which lends more confidence in the interpretation that the observed correlation between aerosol viral load and the number of particles of 1–2.5 μm is related to bioaerosol emissions. These results provide further evidence of the importance of

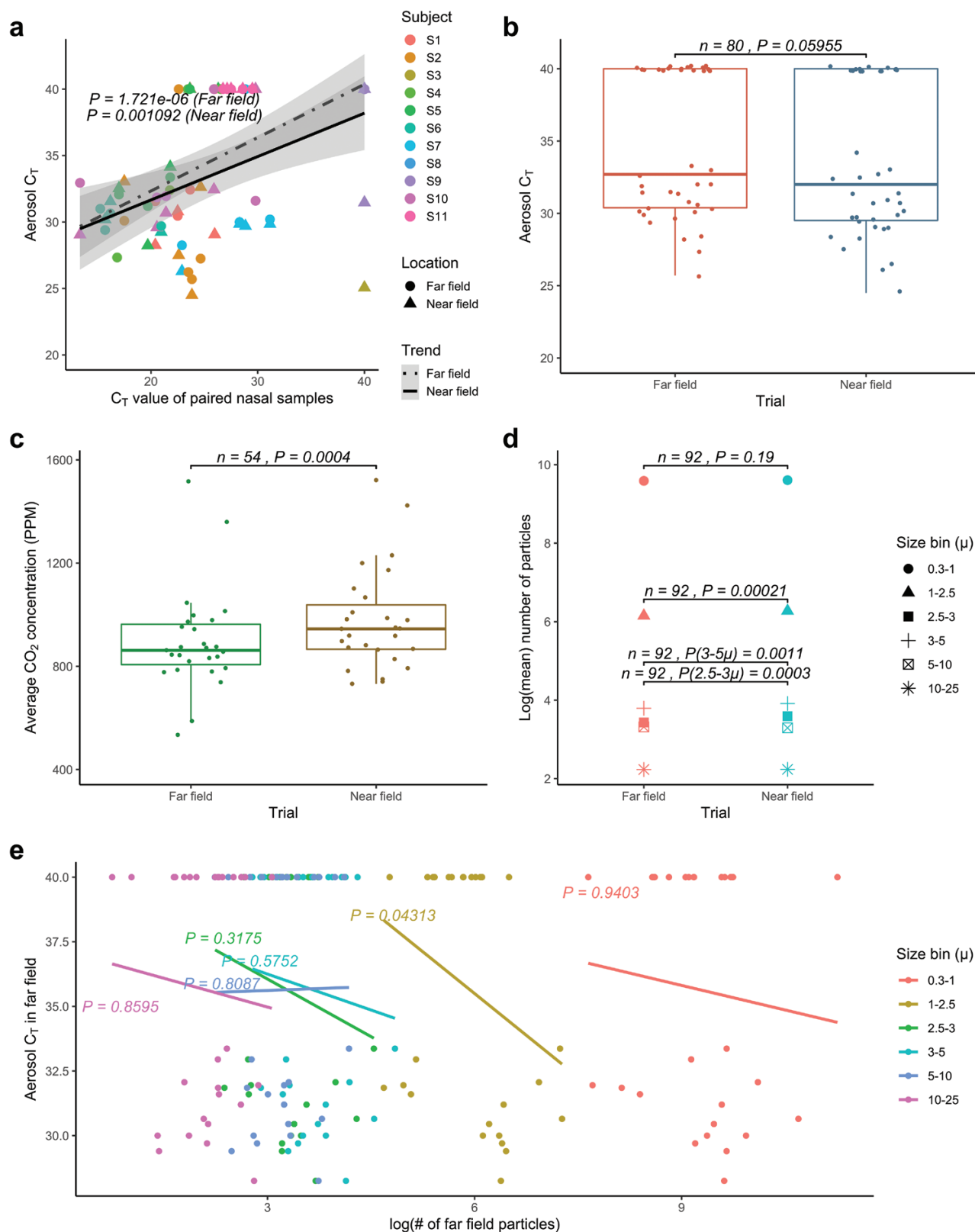


Figure 2. A, Correlation of near field (1.2 m) or far field (3.5 m) aerosol viral loads (RNA) with corresponding human nasal samples during routine trials. B, Comparison of near field and far field aerosol viral loads for routine trials. C, Comparison of mean carbon dioxide concentrations in the near field and far field for routine trials. D, Paired t tests for all particle size bins at near field and far field for routine trials. E, Correlation between mean far field aerosol viral loads and the corresponding mean concentration of far field particles for routine trials. Abbreviations: CO_2 , carbon dioxide; C_T , cycle threshold; PPM, parts per million.

fine aerosols in the potential for COVID-19 disease transmission in both near and far fields.

High-Touch Surfaces, Settling Plates, and Paired Human Specimens

Human specimens were compared to paired samples collected from the participants' phone (screen), computer (adjacent to keyboard), and chair (described as high-touch surfaces), and from near field settling plates (on participant's desk) and far field plates (adjacent to far field air sampler). **Figure 3A** illustrates the significant linear regressions for the viral load (RNA) on each high-touch surface relative to paired nasal samples. **Figure 3B** illustrates the significant linear regressions for viral load in settling plates (near and far) relative to paired nasal samples. There are no significant differences between the viral loads found in near field and far field setting plates, nor are there significant differences between any of the high-touch surfaces (**Supplementary Figures 4 and 5**). **Figure 3C** illustrates the significant regressions for all sampling types relative to human

nasal samples within a single figure and indicate that high-touch surfaces and aerosol samples have stronger correlations to human viral loads than settling plate surfaces. This lends more evidence that emitted virions are present in indoor spaces within smaller particles that remain as aerosols for long time periods.

High Expiratory Activity, Particles, and Aerosol Viral Load

We found a significant correlation between aerosol viral load associated with high expiratory activities and paired nasal samples where an increase in viral load equivalent to $-1 C_T$ in human nasal samples is associated with increased immediate field (<1 m; **Figure 1A**) aerosol viral loads as follows: $-0.189 C_T$ ($R^2 = 0.09058$, $P = .0225$) for 1-minute cough tests, $-0.271 C_T$ ($R^2 = 0.1979$, $P = .00115$) for 5-minute speaking tests, and $-0.229 C_T$ ($R^2 = 0.1796$, $P = .00141$) for 5-minute speaking loudly tests (**Supplementary Figure 6**). Furthermore, we found a significant positive relationship between the mean number

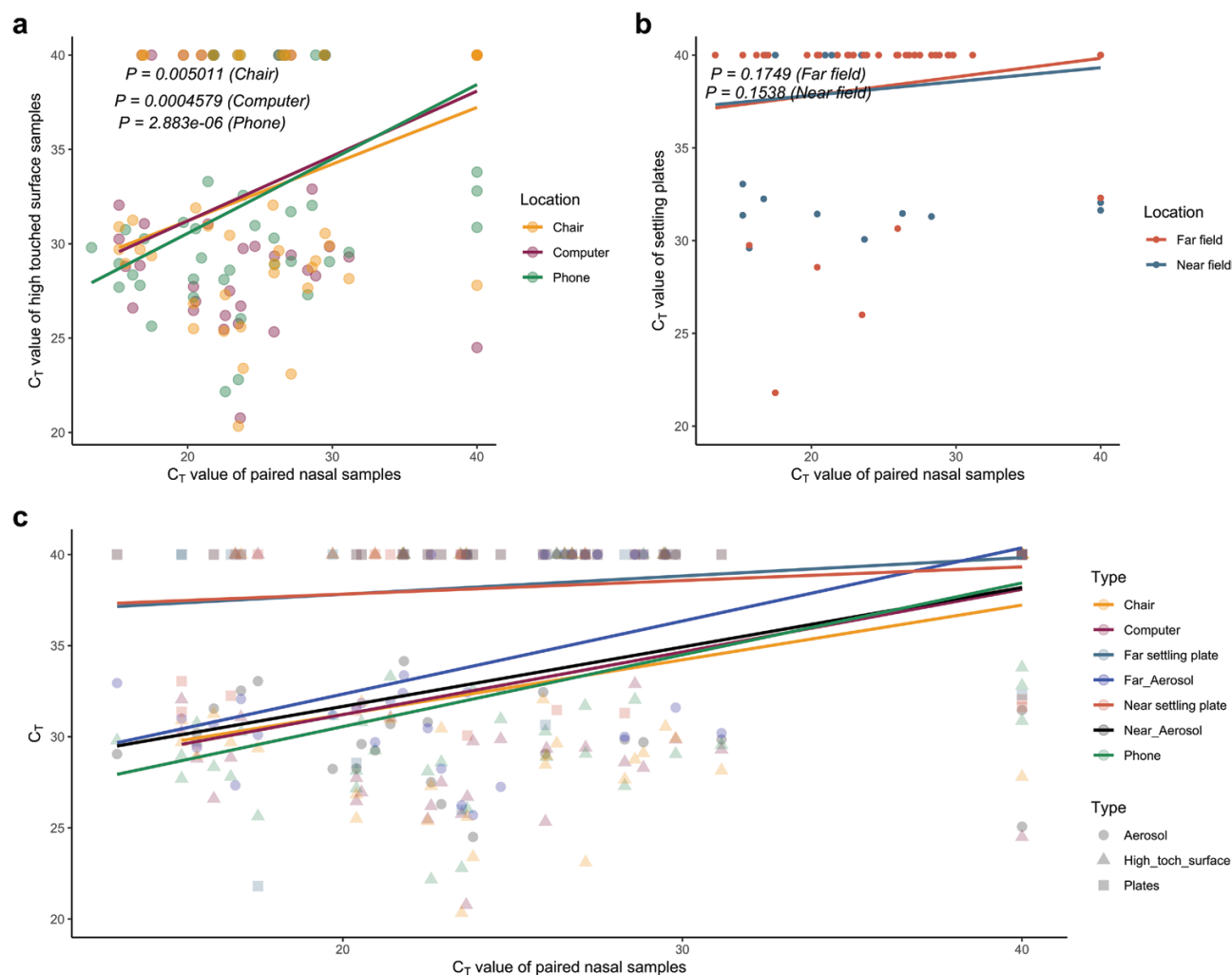


Figure 3. A, Viral load (RNA) on each high-touch surface relative to paired nasal samples. B, Viral load (RNA) on settling plates at near and far field relative to paired nasal samples. C, Correlation of each sample type (aerosol, high-touch surfaces, and settling plates) to paired nasal sample. Abbreviation: C_T, cycle threshold.

of immediate field particles during high expiratory activities (Setup 1) in the size ranges 0.3–1 μm (Figure 4A), 1–2.5 μm (Figure 4B), and 10–25 μm (Figure 4E) and the viral load in

the immediate field aerosols, while the other particle size bins are not significant (Figure 4). We provided further analysis of the relationship between different respiratory activities and

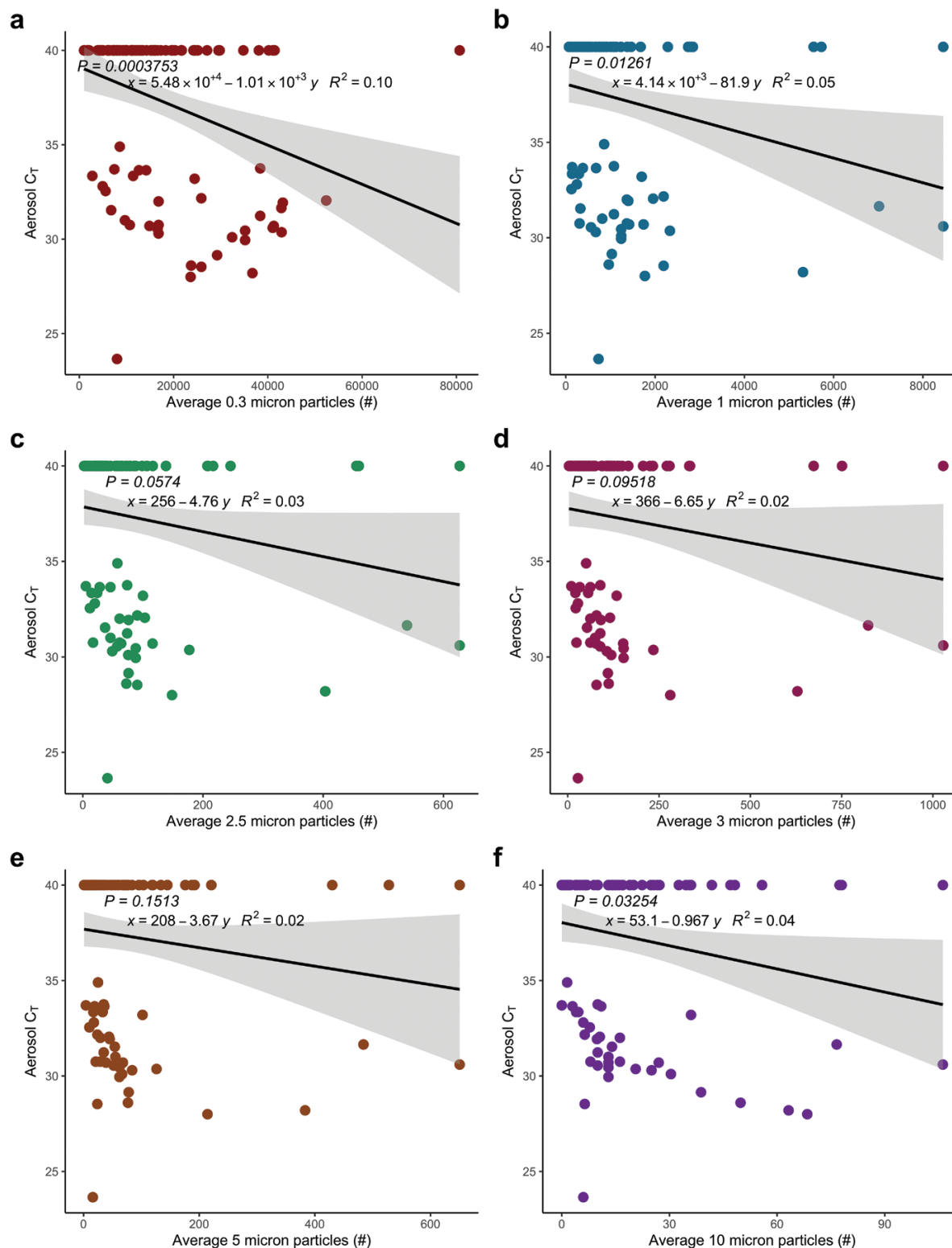


Figure 4. Linear correlation between cycle threshold value and particle size for 0.3–1 μm particles (A), 1–2.5 μm particles (B), 2.5–3 μm particles (C), 3–5 μm particles (D), 5–10 μm particles (E), and 10–25 μm particles (F). Abbreviation: Ct, cycle threshold.

viral loads in [Supplementary Figures 7 and 8](#). Further discussion about the relationship between aerosol viral loads and particles of different size bins are provided in the [Supplementary Document, Appendix C](#).

Impact of Ventilation and Filtration on Aerosol and Surface Viral Load

Indoor air exchange rate, measured in ACH, has previously been demonstrated to reduce indoor particles and therefore hypothesized to reduce the concentration of viral aerosols, corresponding inhalation dose, and consequently the probability of indoor occupants acquiring infection [16, 32–34]. Few studies have measured the relationship between ventilation, filtration, and aerosol viral load [35]. Therefore, we investigated the impact of alternate air exchange rates, using 100% outside air (OSA) and filtration levels during removal mechanism trials. As shown in [Table 1](#), each removal mechanism day began with a baseline ~ 0 ACH trial, followed by 4 100% OSA ventilation trials (2 at ~ 9 ACH and 2 at ~ 3 – 4.5 ACH) provided by an exhaust fan (fitted with high-efficiency particulate air [HEPA] filter for infection control). Thereafter, a single trial with 2 in-room HEPA filters (without OSA) was conducted. All removal mechanism trials and the ~ 0 ACH control trials were conducted for a duration of 1 hour. We found a significant difference between control trials and all removal mechanism trials ($P = .029$; [Figure 5A](#)). In [Figure 5A](#) we show a significant difference between control trials and paired removal mechanism trials, whereas in [Figure 5B](#) we show a significant correlation for all control trials at approximately 0 ACH and all ventilation trials with 100% OA organized by mean CO_2 concentration. Trials with less than ~ 4.5 ACH (including ~ 0 ACH trials) were associated with significantly higher aerosol viral loads in the near field when compared with trials greater than ~ 9 ACH, with a mean difference of $-3.6 C_T$ ($P = .037$, unpaired t test; [Figure 5C](#)). Even though the mean difference of aerosol viral load in the far field for trials with less than ~ 4.5 ACH (including ~ 0 ACH trials) was higher than trials with greater than ~ 9 ACH, we did not observe a statistically significant difference for far field aerosol viral load ($P = .085$, unpaired t test, [Figure 5C](#)). When examining total room aerosol viral load (near field

and far field together), we report that trials with less than ~ 4.5 ACH (including ~ 0 ACH trials) were associated with statistically higher viral load than trials with greater than ~ 9 ACH, with a mean difference of $-3.2 C_T$ ($P = .01153$, unpaired t test; [Supplementary Figure 9](#)). Our research provides further evidence that improved ventilation and filtration is beneficial for both near field and far field aerosol viral load ([Supplementary Table 2](#)). Given these relationships within this room ([Figure 5B](#)), ventilation trials indicate that an increase in ~ 128 PPM of CO_2 concentration corresponds with an increase in aerosol viral load equivalent to $-1 C_T$, thus, approximately a doubling of the viral load. Moreover, filtration trials indicate that there is a significant difference between trials with only in-room HEPA filtration ($\sim 1000 \text{ m}^3/\text{hour}$) and paired control trials at ~ 0 ACH, where HEPA trials have lower viral load equivalent to $3.240741 C_T$ ($P = .029$), thus, approximately an order of magnitude reduction ([Figure 5D](#)).

Our results provide evidence that increased air exchange (~ 9 ACH with 100% OSA) or in-room HEPA filtration ($\sim 1000 \text{ m}^3/\text{hour}$) yields reduced aerosol viral load, and reason therefore suggests these measures are likely to reduce inhalation dose and the probability of infection in indoor spaces. We found no statistical difference between aerosols captured during control trials with ~ 0 ACH and those with ~ 3 – 4.5 ACH; however, this may be related to limitations in sample size. Among 3 types of high-touch surfaces collected in this study, increased ACH was associated with lower viral load on participants' computers, with a mean difference of $4.033 C_T$ ($P = .002323$), whereas phone and chair samples showed no significant difference with air exchange rate ([Supplementary Figure 10](#)).

Relative Humidity and Aerosol Viral Load

Relative humidity (RH) is hypothesized to impact aerosol pathogens and disease transmission in 3 ways: (1) improved human immune response [33]; (2) reduced viability in aerosols at RH between 40% and 60% [15, 23]; and (3) increased particle deposition [16, 36]. The structure and behavior of aerosol pathogens, specifically particle size, settling rate, and diffusion, are each affected by RH [36, 37]. In this study, we aimed to

Table 1. Study Plan for Participants Who Were Diagnosed With Coronavirus Disease 2019

Setup	Physical Activity	Removal Mechanism	Relative Humidity
S1	10 coughs in 1 minute	10 coughs in 1 minute	10 coughs in 1 minute
S1	Speak for 5 minutes	Speak for 5 minutes	Speak for 5 minutes
S1	Speak loudly for 5 minutes	Speak loudly for 5 minutes	Speak loudly for 5 minutes
S2	1 hour regular sitting	1 hour regular sitting	1 hour regular sitting
S2	1 hour standing	1 hour sitting at ~ 9 ACH	1 hour sitting at low RH
S2	30 minutes sitting silently	1 hour sitting at ~ 3 ACH	1 hour sitting at low RH
S2	30 minutes sitting speaking	1 hour sitting at ~ 9 ACH	1 hour sitting at high RH
S2	15 minutes walking on treadmill	1 hour sitting at ~ 4.5 ACH	1 hour sitting at high RH
S2	...	1 hour sitting with HEPA filtration	...

Abbreviations: ACH, air changes per hour; HEPA, high-efficiency particulate air; RH, relative humidity; S1, experimental setup 1; S2, experimental setup 2.

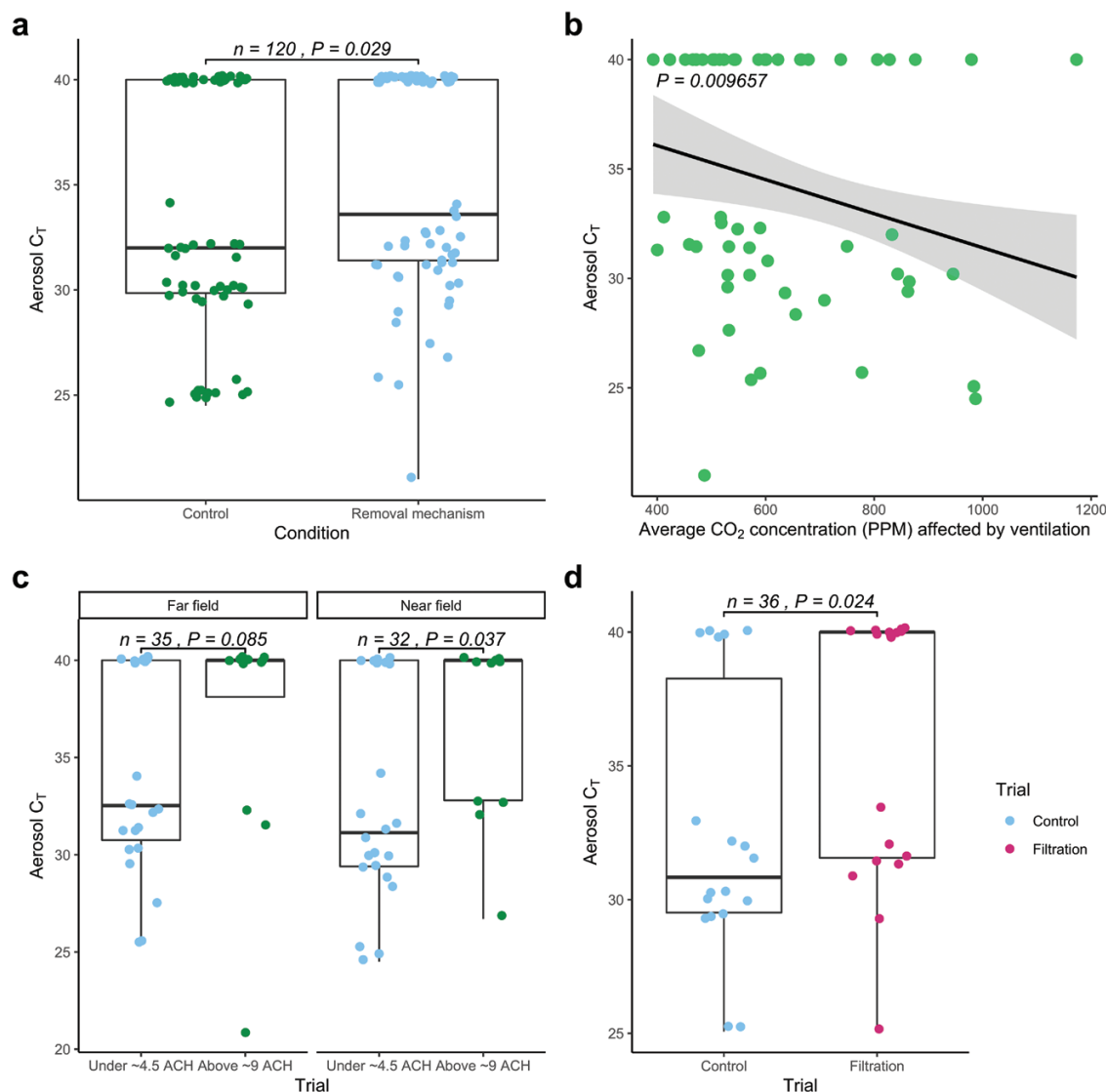


Figure 5. The impact of ventilation and filtration on cycle threshold (C_T) value of aerosol samples. *A*, Matched paired comparison between trials with removal mechanism trials (filtration and ventilation) and control trials with ~0 ACH. *B*, Linear correlation between aerosol C_T value and paired mean carbon dioxide concentration affected by only ventilation (same physical activities). *C*, Comparison of aerosol C_T for ventilation trials of under ~4.5 air changes per hour (ACH) and above ~9 ACH in near field and far field. *D*, Matched paired comparison of aerosol C_T for trials with in-room high-efficiency particular air filtration and corresponding control trials with ~0 ACH. Abbreviations: ACH, air changes per hour; CO₂, carbon dioxide; C_T, cycle threshold; PPM, parts per million.

measure environmental viral load at different RH conditions. Two dehumidifiers and 2 humidifiers were used to regulate RH to low (22.2%–38.9%; mean = 28.8%) and high (44.83%–61%; mean = 53.9%) levels during the “relative humidity” trials. Each participant’s relative humidity day started with a 1-hour control trial with ~0 ACH and RH at ambient conditions, followed by two 1-hour dehumidification trials and two 1-hour humidification trials. Room aerosol C_T values were paired with mean RH values (ranging from 20% to 70%) recorded for each trial.

Relative humidity trials indicate that an increase of ~11.85% in RH corresponds with a decrease in aerosol viral load equivalent to 1 C_T ($P = .008$), thus approximately a 50% reduction in aerosol viral load, as shown in Figure 6A. Similarly, an increase

of ~10.02% in RH corresponds with an increase in surface (chair, computer, phone) viral load equivalent to -1 C_T ($P = .01$) as shown in Figure 6C, consistent with increased particle deposition. Figure 6B shows the significant decrease in aerosol viral load equivalent to 3.289 C_T (paired t test, $P = .0002643$) for humidification trials as compared to dehumidification trials. Conversely, Figure 6D shows the significant increase in computer surface viral load equivalent to -2.873 C_T (paired t test, $P = .01593$) for humidification trials as compared to dehumidification trials.

This is one of the first studies that investigated the role of RH on viral RNA in aerosols and surfaces in a realistic setting. Our results suggest that increased RH corresponds with decreased viral load in aerosols and increased viral load on select

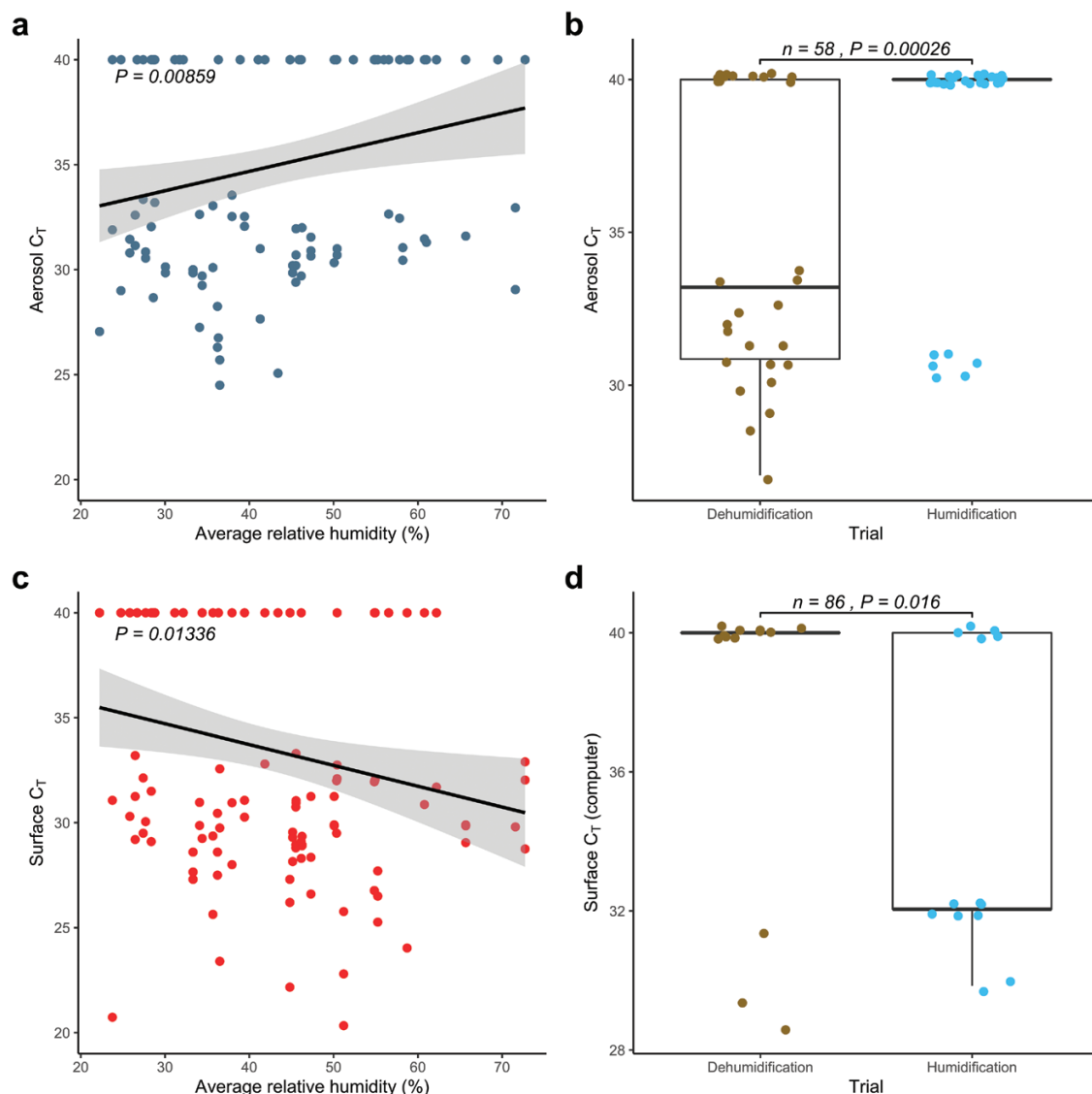


Figure 6. A, Correlation between aerosol cycle threshold (C_t) value and mean relative humidity among dehumidification, humidification, and control trials. B, Paired comparison of aerosol C_t between dehumidification and humidification trials. C, Correlation between surface C_t value and mean relative humidity among dehumidification, humidification, and control trials. D, Paired comparison of select surface (computer) C_t between dehumidification and humidification trials. Abbreviation: C_t , cycle threshold.

indoor surfaces, consistent with an increased rate of particle deposition. Since several studies have demonstrated that there is a substantially higher risk for aerosol-mediated transmission than fomite-mediated transmission [38], active humidity control (including humidification, or reduced dehumidification) could be implemented to reduce aerosol-mediated COVID-19 transmission risk reduction in indoor spaces. Of course, humidification controls must be properly maintained and managed to avoid condensation and mold propagation.

DISCUSSION

There were several limitations to this study. All participants were given the opportunity to opt out of the study at any time, and 2 subjects only completed the first day of study. There

were some modest inconsistencies between trial durations in order to accommodate participants' needs. Not all participants walked on the treadmill, and some walked at different speeds or for different durations. Participants may have presented inconsistent symptoms (such as coughing) during the course of the experiments; however, the control trial at the beginning of each day addresses a substantial part of this limitation. While this was an extensive study design, conducted over 3 days per participant (Supplementary Figures 11 and 12), the total number of unique participants ($n = 11$) and limited age range (18–24 years of age) of participants presents some limitations to generalizability. RNA samples were not assessed for viability.

In summary, we found the following statistically significant relationships:

1. Positive relationships between viral load (RNA) found in human specimens and paired aerosol and surface samples at ~0 ACH and ambient conditions for sitting and standing trials (routine trials) as well as trials with high expiratory activities (coughing, speaking, and speaking loudly).
2. Positive relationship between viral load in near field aerosols captured during periods of higher expiratory activity and near field particles of 0.3–1 μm , 1–2.5 μm , and 10–25 μm in size, but no statistical significance for 2.5–10 μm particles.
3. Increased CO_2 concentrations and particle counts in the range of 1–5 μm measured in the near field as compared to the far field for routine trials.
4. Positive relationship between aerosol viral load in the far field and the number of corresponding far field particles detected in the range of 1–2.5 μm .
5. Inverse relationships between viral load found in aerosols and degree of ventilation, as well as in-room filtration.
6. Relationships between viral load and degree of relative humidity; whereby higher RH is associated with lower viral load in aerosol samples and higher viral load in select surface samples, consistent with increased particle deposition on surfaces.

Notes

Author Contributions. K. G. V. D. W. performed funding acquisition and managed the investigation team. K. G. V. D. W. and H. P. conceived of the project scope and methodology with input from L. D. and P. F. H., and K. G. V. D. W. and L. D. enrolled and consented study participants. D. N. worked with all other authors to set up modular room and all research instrumentation. H. P., L. B., L. D., and A. O. M. collected biological specimens. L. B., L. D., P. F. H., and A. O. M. performed laboratory analyses. H. P. performed data curation and data exploration, developed final analysis scripts, performed final analysis, and created visualizations with support from all authors. H. P. developed the original manuscript with direction and input from K. G. V. D. W. All authors provided manuscript revisions and edits on subsequent manuscript drafts and approved the final manuscript.

Acknowledgments. The authors thank all staff in the BioBE Center, including Garis Bowles, Georgia MacCrone, Jackson Mestler, Dan Richards, and Vincent Moore. The authors would like to thank countless individuals at the University of Oregon for helping to make this research design possible. The authors would also like to thank Dr Richard Corsi, Dr Siobhan Rockcastle, and Dr Mark Fretz for their input during results analysis. Most importantly, we would like to thank all study participants for their commitment during a difficult period in their lives. All data and code supporting this study and required to create the analyses are provided in Github, available at <https://github.com/BioBE/RDM>.

Disclaimer. The funder provided the university with support in the form of salaries for all authors, equipment, and reagents but did not have any additional role in the study design, data collection and analysis, decision to publish, or preparation of the manuscript. However, per contractual obligations, the funder had the right to review the final manuscript for confidential information prior to submission.

Financial support. This work was supported by Thermo Fisher Scientific (award number 4133V1).

Potential conflicts of interest. K. G. V. D. W. has a company called Duktile that provides consulting related to healthy buildings and pathogen control; serves as a scientific advisor to EnviralTech and Poppy, companies that conduct viral environmental surveillance and infection control; serves as scientific advisor to the Integrated Bioscience and Built Environment Consortium; and reports honorarium for a lecture about

building mitigation strategies inclusive of ≥ 5 papers written by our research team from Pacific Gas & Electric. P. F. H. serves as Director of Resource Archiving for the Microbes and Social Equity Working Group. L. D. serves as Eugene chapter lead for the 500 Women Scientists group. L. G. D. reports being Chapter Leader of 500 Women Scientists (unpaid). All other authors report no potential conflicts of interest.

All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

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