

Longitudinal Analysis of Built Environment and Aerosol Contamination Associated with Isolated COVID-19 Positive Individuals

Patrick Horve

Biology and the Built Environment Center, University of Oregon, Eugene, OR, 97403

Leslie Dietz

Biology and the Built Environment Center, University of Oregon, Eugene, OR, 97403

Garis Bowles

Biology and the Built Environment Center, University of Oregon, Eugene, OR, 97403

Georgia MacCrone

Biology and the Built Environment Center, University of Oregon, Eugene, OR, 97403

Andreas Olsen-Martinez

Biology and the Built Environment Center, University of Oregon, Eugene, OR, 97403

Dale Northcutt

Energy Studies in Buildings Laboratory, University of Oregon, Eugene, OR, 97403

Vincent Moore

Biology and the Built Environment Center, University of Oregon, Eugene, OR, 97403

Liliana Barnatan

Biology and the Built Environment Center, University of Oregon, Eugene, OR, 97403

Hooman Parhizkar

Energy Studies in Buildings Laboratory, University of Oregon, Eugene, OR, 97403

Kevin Van Den Wymelenberg (✉ kevinvdw@uoregon.edu)

Institute for Health and the Built Environment, University of Oregon, Portland, OR, 97209

Research Article

Keywords: COVID-19, SARS-CoV-2, aerosol, viral load

DOI: <https://doi.org/10.21203/rs.3.rs-861942/v1>

License:   This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

1 **Title: Longitudinal Analysis of Built Environment and Aerosol Contamination Associated**
2 **with Isolated COVID-19 Positive Individuals**

3
4 **Funding Statement:** Funding for this research was partially provided by Thermo Fisher Scientific
5 under award number 4133V1, as well as a confidential gift to the University of Oregon.

20 **Abstract**

21 The indoor environment is the primary location for the transmission of severe acute respiratory
22 syndrome coronavirus 2 (SARS-CoV-2), the causative agent of coronavirus disease 2019 (COVID-
23 19), largely driven by respiratory particle accumulation in the air and increased connectivity between
24 the individuals occupying indoor spaces. In this study, we aimed to track a cohort of subjects as they
25 occupied a COVID-19 isolation dormitory to better understand the impact of subject and
26 environmental viral load over time, symptoms, and room ventilation on the detectable viral load
27 within a single room. We find that subject samples demonstrate a decrease in overall viral load over
28 time, symptoms significantly impact environmental viral load, and we provide the first real-world
29 evidence for decreased aerosol SARS-CoV-2 load with increasing ventilation, both from mechanical
30 and window sources. These results may guide environmental viral surveillance strategies and be used
31 to better control the spread of SARS-CoV-2 within built environments and better protect those
32 caring for individuals with COVID-19.

33

34

35

36

37

38

39 **Introduction**

40 The built environment (BE)^{1,2}, or the spaces that we, as humans, have built for ourselves to work in,
41 inhabit, and enjoy life, play an essential role in mitigating the spread of severe acute respiratory
42 syndrome coronavirus 2 (SARS-CoV-2), the causative agent of coronavirus disease 2019 (COVID-
43 19)³. SARS-CoV-2 transmission indoors is aided through extended close contact and the
44 accumulation and persistence of aerosolized SARS-CoV-2, largely driven by poor ventilation⁴⁻¹⁷.
45 Significant effort has gone into the identification of SARS-CoV-2 in a multitude of BE's^{6,8,18,19,20,20-32}.
46 However, most efforts to understand the environmental contamination associated with individuals
47 diagnosed with COVID-19 have been performed at a single time point, missing critical information
48 about the longitudinal dynamics of that environmental contamination. Additionally, minimal
49 characterization has been performed to understand how symptoms and BE factors such as
50 ventilation, measured in air changes per hour (ACH), impact the total environmental and aerosolized
51 contamination by SARS-CoV-2 within the BE over time.

52 One common scenario faced by people throughout the world is co-occupation of an indoor space
53 with a COVID-19 positive individual while they themselves are not known to be positive. Three of
54 the major outstanding questions in regard to COVID-19 infections and the built environment
55 include (i) how individuals emit virus into the environment over time, (ii) how different forms of
56 environmental sampling are able to support biosurveillance initiatives, and (iii) and to what degree
57 does ventilation mitigate environmental contamination. In order to better understand the
58 longitudinal dynamics associated with the occupation of the BE when suffering from COVID-19,
59 the impact of ventilation, and the potential role of different surveillance methods, isolation dorm
60 rooms housing residence hall students that tested positive for COVID-19 were sampled throughout
61 the course of the individual's isolation period, typically allowing for up to 10 days of sample
62 collection. Here, we provide the first real-world experimental evidence for the suppression of

63 aerosol viral loads through the use of increased ACH from exhaust air and increased natural
64 ventilation through the use of windows. Additionally, we demonstrate that symptom type, severity,
65 and presence are predictive factors for the level of environmental contamination observed and that
66 environmental contamination decreases as individuals recover. Additionally, we identified variability
67 in viral shedding over time and provide evidence useful to guide environmental viral surveillance.

68 **Results and Discussion**

69 **Study Population**

70 A total of 35 subjects were recruited and consented into the study between January and May 2021.
71 All subjects tested positive for SARS-CoV-2 RNA through shallow nasal swabs and qRT-PCR. The
72 study cohort was made up of 17 males and 18 females between the age of 18 and 24 (Table 1). The
73 majority of individuals in the study cohort identified as White (68.6%) followed by
74 Hispanic/Latino/Spanish (14.3%). A full breakdown of the self-identifying ethnicity of the study
75 cohort can be found in Table 1.

76 **Table 1. Demographic data of the study subjects.**

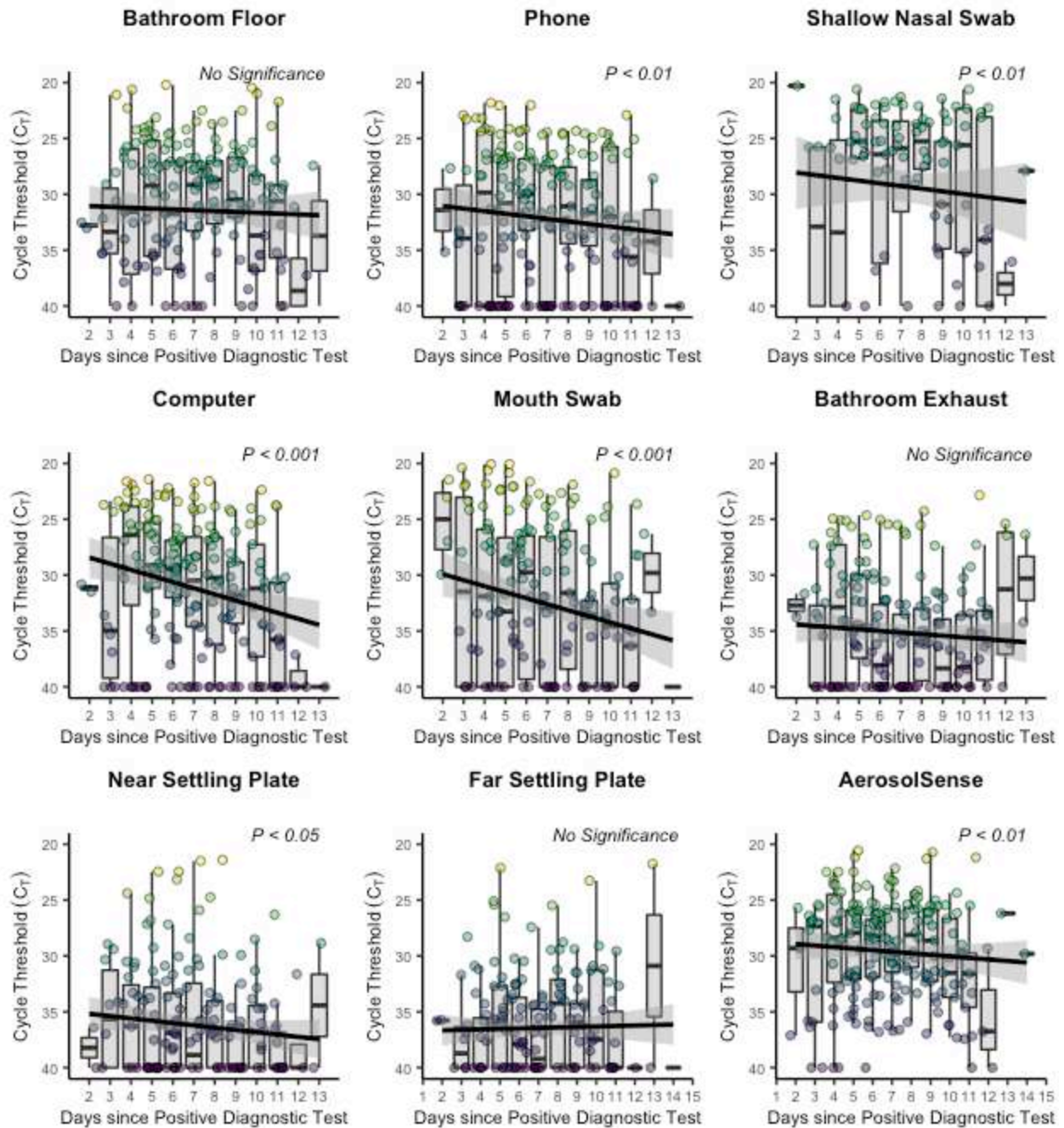
Sex at Birth	Percent (n)
Male	48.6 (17)
Female	51.4 (18)
Ethnicity	Percent (n)
White	68.6 (24)
Hispanic/Latino/Spanish	14.3 (5)
Native Hawaiian/Pacific Islander	2.9 (1)
Black	2.9 (1)
Multiple	5.7 (2)
Asian	5.7 (2)

77 *Viral Shedding and Environmental Contamination Associate with Isolation Day*

78 In an attempt to assess the viral load dynamics over the course of the study cohort's time in the
79 isolation dormitory, the mean C_T , a proxy for observed total viral load, of each study participant
80 from each location was tracked throughout the course of the isolation period. C_T values of subject
81 shallow nasal and mouth swabs were found to be significantly ($P < 0.05$) associated with day since
82 positive test, with C_T values increasing (lower viral load) as time since positive test increases (Figure
83 1). Additionally, significant increases in C_T values were observed as time progressed in environmental
84 swabs taken from the study subject's computer, phone, the settling plate closest to the study
85 participant, and in the active air samples (AerosolSense). Statistically significant increases in the C_T
86 values of participant bathroom floors, bathroom exhaust, and far passive air settling plate were not
87 observed, although nearly all sample types trended towards increased C_T values over time.
88 Furthermore, environmental samples demonstrated decreasing percent positivity over time (Figure
89 2).

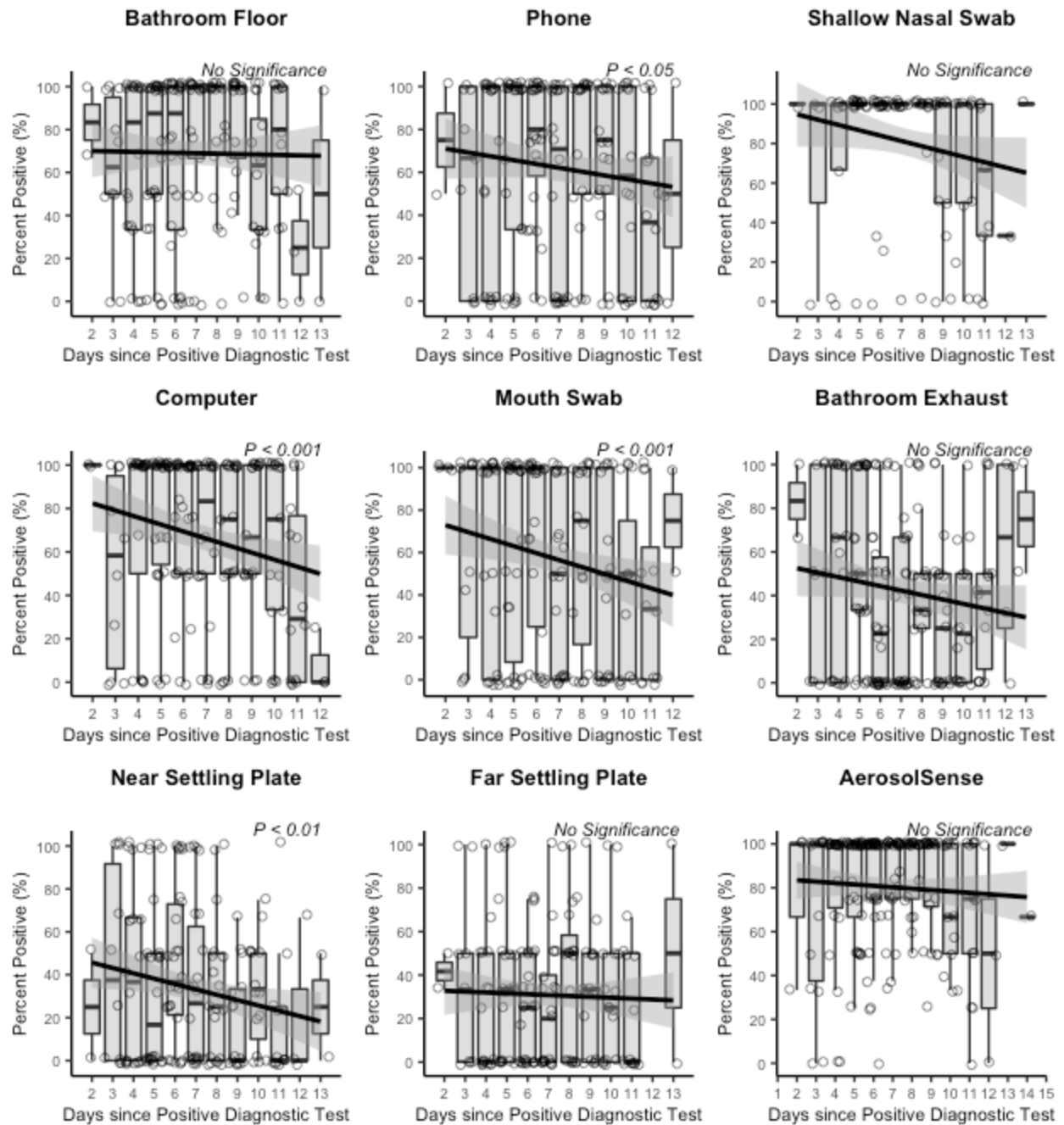
90 Increasing nasal and mouth C_T values and decreasing rate of positivity of environmental samples as
91 the isolation period progresses both suggest that decreasing viral load in study participants directly
92 translates to decreased viral load within the space occupied by individuals positive for COVID-19
93 (Figure 1). While previous investigations have demonstrated the presence of SARS-CoV-2 RNA in
94 BE's occupied by COVID-19 positive individuals^{5,18,23,32-34}, this represents the first link between
95 infection stage, subject viral load over time, and environmental viral load. Additionally, we confirm
96 the findings of multiple other studies that have demonstrated the persistence of SARS-CoV-2
97 genetic material in patient-derived samples at the end of a treatment and/or isolation period^{35,36}. The
98 persistence of environmental SARS-CoV-2 genomic material has been cited as a potential limitation
99 in multiple sampling campaigns that utilize surface swabs to assess contamination^{37,38}. The strongest
100 trends in increasing C_T values among environmental samples were observed in the phone and

101 computer swabs, and AerosolSense active air samples. In comparison to samples that did not
102 demonstrate a significant increase in C_T values over time (bathroom exhaust and bathroom floor),
103 these sampling locations were either cleaned in between sampling (phone and computer) or utilize a
104 fresh substrate during each collection period (AerosolSense). This comparison suggests that relic
105 RNA may compose at least a part of the RNA collected in some environmental surveillance
106 sampling methods and that sampling methods that routinely cleaned or were more resistant to relic
107 RNA collection (such as active air sampling with a fresh substrate) may provide more utility as a
108 surveillance tool against SARS-CoV-2 than typical environmental swabbing campaigns.



109

110 Figure 1. Longitudinal Viral Shedding and Environmental Contamination Dynamics. The mean daily cycle
 111 threshold (C_T) for each sampling location throughout the course of the participants' involvement in the study.
 112 Individual points represent the mean daily C_T value per individual. The black line represents a linear mixed
 113 model estimated using a restricted maximum likelihood (REML) approach and including the individual
 114 occupying the room as a random effect and the grey area represents the 95% confidence interval for that
 115 model.



116

117 **Figure 2.** Mean daily percent positivity at each sampling location. The percent positivity rate per entry per
 118 study subject was calculated and the mean positivity rate of all participants per day enrolled in the study was
 119 calculated as the daily percentage rate. The black line represents a linear mixed model estimated using a
 120 restricted maximum likelihood (REML) approach and including the individual occupying the room as a
 121 random effect and the grey area represents the 95% confidence interval for that model.

122 *Symptom Presence Impacts Viral Shedding and Environmental Contamination*

123 The presence (or lack thereof) of symptoms associated with COVID-19 positive individuals and

124 associated viral load in patient samples (nasopharyngeal and oral swabs) has been investigated in a

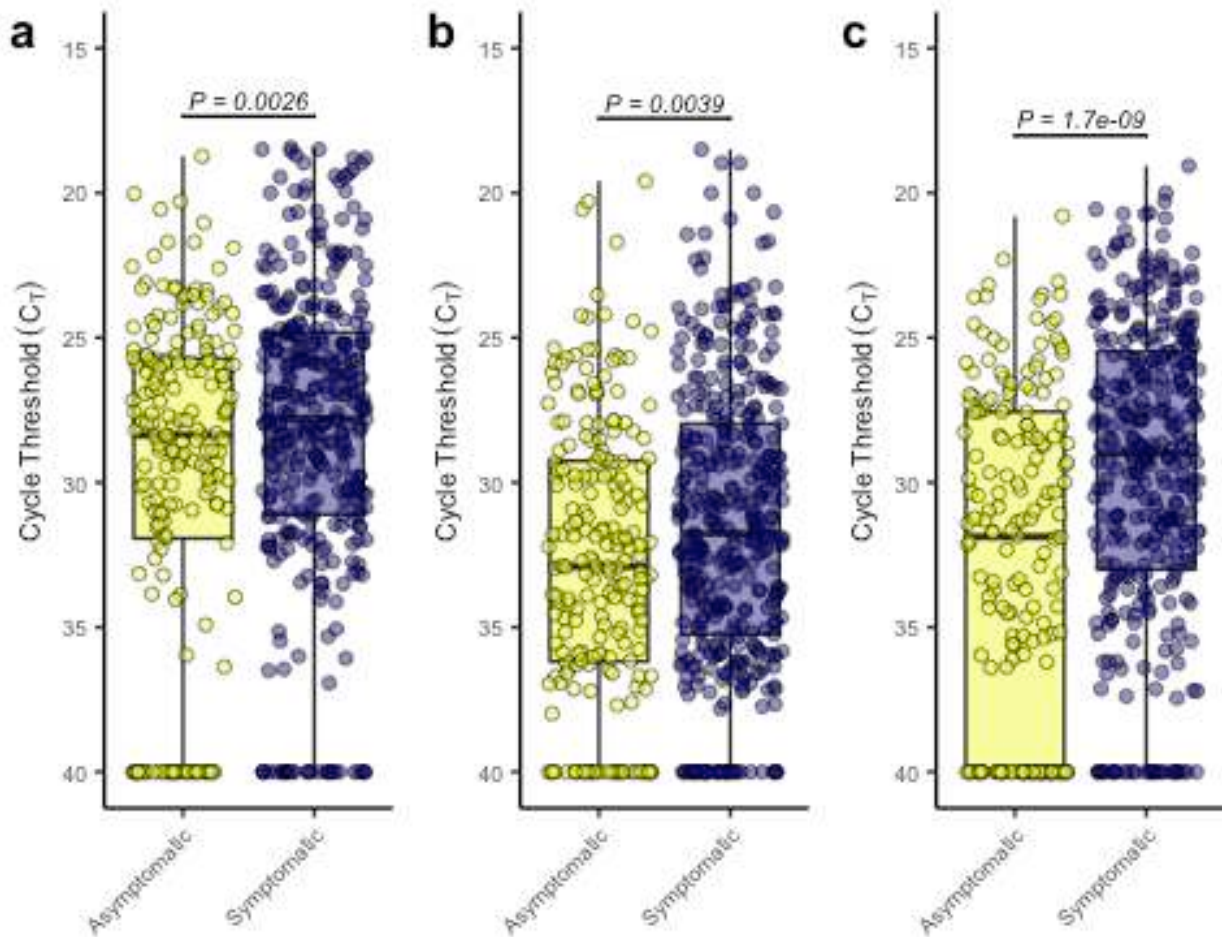
125 multitude of previous articles and significant differences have not been identified in the viral load
126 associated with symptomatic versus asymptomatic COVID-19 infections³⁹⁻⁴⁷. However, the
127 relationship between symptomatic infection and environmental contamination has not yet been
128 investigated. Among the symptoms that were reported by the study population, seven symptoms
129 (coughing, watering eyes, sore throat, loss of smell, gastrointestinal (GI) symptoms, congestion, and
130 brain fog) were found to be significantly associated with altered levels of viral load in the isolation
131 dormitory rooms (Table 2). Increased self-reported coughing, sore throat, loss of smell, and GI
132 symptoms were associated with lower environmental C_T values (and thus higher viral loads), with GI
133 symptoms and coughing most strongly correlating with decreased C_T values. In comparison, watery
134 eyes, congestion, and brain fog were associated with increased C_T values. Coughing while infected
135 with COVID-19 has been estimated to produce significantly more viral particles than normal
136 breathing⁴⁸. This small cohort study of 35 individuals supports the hypothesis that increased
137 respiratory expulsion from activities such as coughing would result in increased environmental
138 contamination with SARS-CoV-2^{49,50}. Furthermore, it is known, through wastewater analysis and
139 sequencing for the surveillance of SARS-CoV-2⁵¹, that SARS-CoV-2 is readily emitted from and
140 detected in stool samples in nearly half of COVID-19 positive individuals⁵². Here we observe
141 increased viral load associated with increased GI symptoms, further supporting the potential for a
142 fecal-oral transmission route of SARS-CoV-2 in certain circumstances. Additionally, the other
143 symptoms associated with increased environmental viral load (sore throat and loss of smell) both
144 implicate the upper respiratory tract. Active viral replication has been identified in the upper
145 respiratory tract and suggests that ongoing infection and symptom onset in the upper respiratory
146 tract may indicate increased levels of viral secretion and environmental contamination in buildings⁵³.

147 **Table 2. Linear correlations between the self-reported symptoms of study participants and measured cycle**
148 **threshold values in the environmental samples. The statistical significance of the correlation for each symptom**
149 **is noted, and the slope indicates the direction of the relationship whereas negative values indicate increased**
150 **environmental viral load.**

Symptom Correlation Coefficients		
Symptom	Slope	Significance Level
Fever	-0.35	Not Significant
Coughing	-0.52	< 0.001
Sneezing	-0.12	Not Significant
Difficulty Breathing	-0.03	Not Significant
Fatigue	0.13	Not Significant
Headache	-0.16	Not Significant
Eyes Ache	0.15	Not Significant
Eyes Watering	1.48	< 0.001
Sore Throat	-0.30	< 0.05
Distorted Taste	0.06	Not Significant
Loss of Taste	0.01	Not Significant
Distorted Smell	0.00	Not Significant
Loss of Smell	-0.13	< 0.01
Ears Ringing	0.37	Not Significant
GI Symptoms	-0.93	< 0.01
Congestion	1.00	< 0.001
Brain Fog	0.31	< 0.01

151 We sought to further understand the potential impact that symptoms play in the transmission of
152 SARS-CoV-2 inside of the BE, and particularly, the impact symptom presence may have on
153 subsequent environmental contamination. As such, each entry into a study participant's room was
154 queried to determine if the participant had self-reported any symptoms during that visit only.
155 Individual entries were sorted into symptomatic and asymptomatic entries and the C_T values from
156 each group were compared. Significantly lower C_T values were observed in active air samples
157 collected during entries where the participant reported symptoms (Figure 3a), representing greater
158 aerosolized viral particles present during that collection time. Furthermore, significantly lower C_T

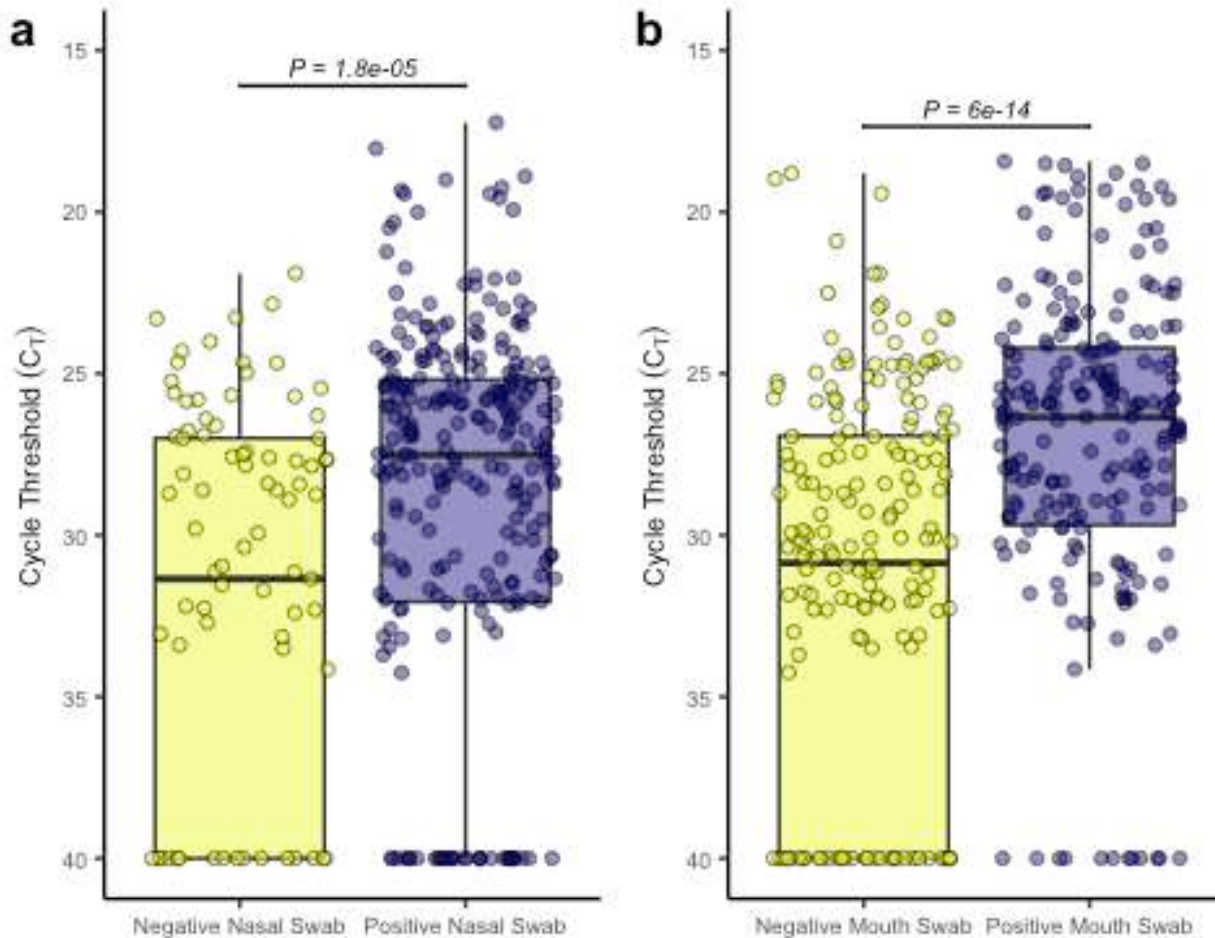
159 values were observed in aerosol-based sampling methods (active air samples and passive settling
160 plates) during symptomatic entries (Figure 3b). Lastly, significantly lower C_T values were also
161 observed in environmental swab samples collected during symptomatic visits compared to
162 asymptomatic visits (Figure 3c). All together, these results suggest the potential that the presence of
163 symptoms, even periodically in some individuals, contributes to increased viral shedding and
164 environmental contamination with SARS-CoV-2.



165

166 **Figure 3. Impact of symptom presence on viral shedding and detection. (a)** Boxplots of the observed cycle
167 threshold values for active air samples collected by the AerosolSense sampler from rooms occupied by
168 asymptomatic (yellow) and symptomatic (purple) individuals. **(b)** Boxplots of observed cycle threshold values
169 for aerosol particulate samples collected by the AerosolSense sampler, passive air settling plate, and bathroom
170 exhaust vents from rooms occupied by asymptomatic (yellow) and symptomatic (purple) individuals. **(c)**
171 **Boxplots of the observed cycle threshold values for environmental swabs collected from the computer, phone,**
172 **and bathroom floor from rooms occupied by asymptomatic (yellow) and symptomatic (purple) individuals.**

173 Additionally, some subjects enrolled in the study demonstrated intermittent negative shallow nasal
174 and oral swabs. To understand whether these intermittent periods of potentially low viral source
175 load further translated to decreased levels of aerosolized viral particles, each entry into a study
176 participant's room was investigated to determine whether a positive or negative human swab
177 (shallow nasal and oral swabs separately) was associated with that entry. Significantly lower C_T values
178 were observed in active air samples collected during entries where the participant returned a positive
179 shallow nasal swab (Figure 4a). This same statistically significant relationship was also observed
180 when grouping samples based upon the result of their oral swabs (Figure 4b). Some intermittent
181 detection of SARS-CoV-2 RNA in the later stages of infection have been previously reported⁵⁴⁻⁵⁶.
182 COVID-19 is unique in that it has been associated with significant numbers of super spreader
183 events^{11,57-59}. It has been suggested that as low as 2% of COVID-19 positive individuals may account
184 for up to 20% of confirmed cases⁵⁷. Here, we find a potential relationship between intermittent
185 positivity, symptom dynamics, and the detectable viral load of the subject and their environment.
186 We hypothesize that individuals suffering from COVID-19 may undergo transient periods of viral
187 shedding that may contribute (among many other factors) to lack of transmission in some exposure
188 events and super spreader transmission in other exposure events. This potential intermittency of
189 viral shedding underscores the value of high temporal resolution of environmental viral surveillance.



190

191 **Figure 4. Potential intermittency of viral shedding and production. (a) Boxplots of the observed cycle**
 192 **threshold values for active air samples collected by the AerosolSense sampler from room entries when the**
 193 **study participant returned a negative shallow nasal swab (yellow) and a positive shallow nasal swab (purple).**
 194 **(b) Boxplots of the observed cycle threshold values for active air samples collected by the AerosolSense**
 195 **sampler from room entries when the study participant returned a negative oral swab (yellow) and a positive**
 196 **oral swab (purple).**

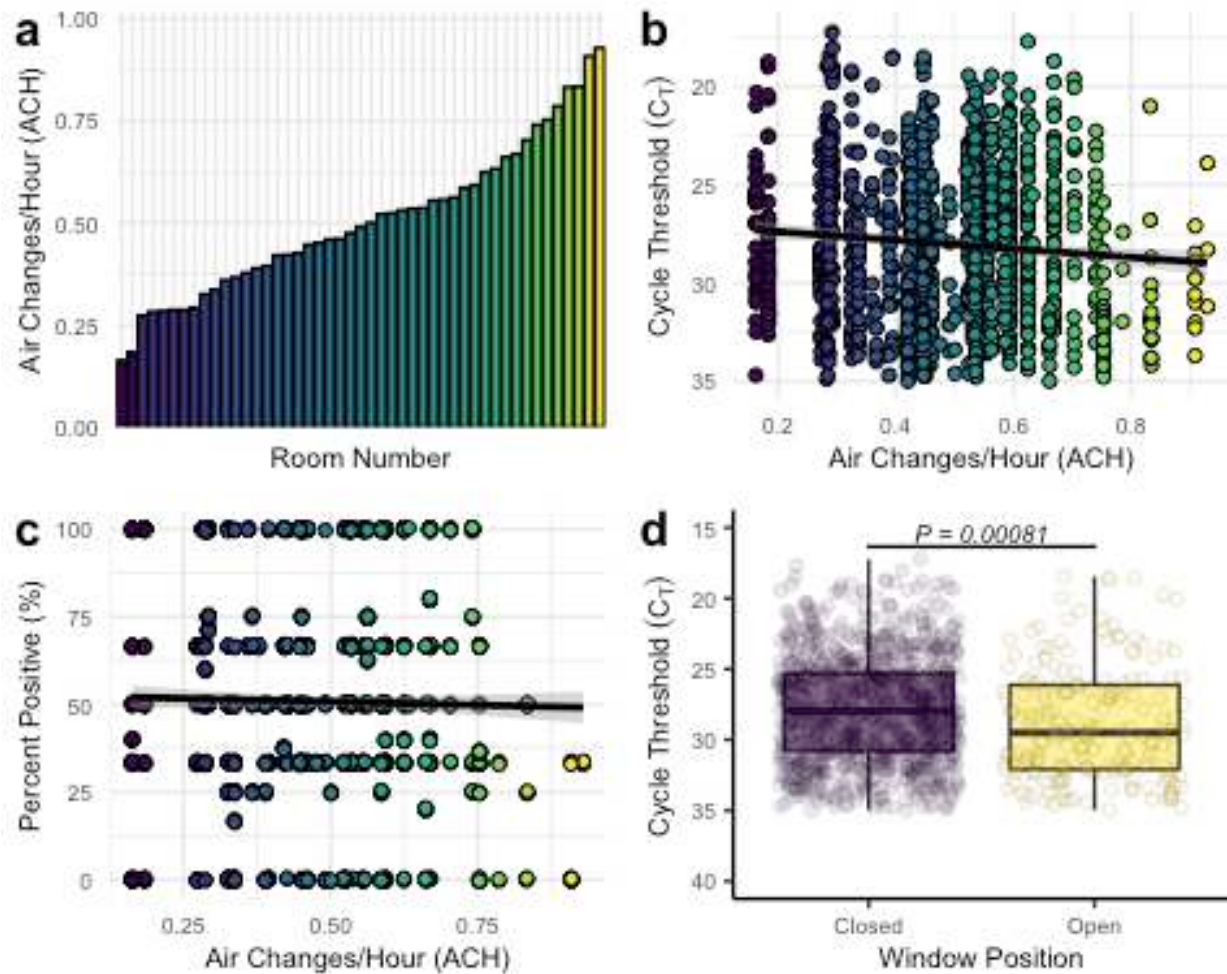
197 *Built Environment Factors and Environmental Viral Detectivity*

198 The BE has been demonstrated to be an area of high risk when there is a COVID-19 positive
 199 individual occupying the indoor space⁶⁰⁻⁶⁴. Despite initial guidance that SARS-CoV-2 is transmitted
 200 through droplets and close interactions between individuals⁶⁵, it has become readily apparent that a
 201 major transmission method is through aerosolized viral particles that remain suspended in the air for
 202 extended periods of time^{5,6,8-10,14-17}. As such, we sought to understand the relationship between a
 203 range of air exchange rates in the isolation rooms studied and detectability of aerosolized SARS-

204 CoV-2. The rate of exhausted air was measured from each isolation dorm room and the air changes
205 per hour (ACH) were calculated for each room (See Materials and Methods for full details). The
206 ACH from mechanically exhausted air in the isolation dorm rooms ranged from 0.16 ACH to 0.93
207 ACH (Figure 5a). Current American Society of Heating, Refrigerating, and Air-Conditioning
208 Engineers (ASHRAE) guidelines suggest a minimum of 0.35 ACH for multifamily units, 1.7 ACH
209 for retail spaces, and 2.8 ACH for classrooms⁶⁶. ACH from mechanical exhaust in the isolation
210 rooms was found to be significantly and positively related to observed C_T values ($P < 0.01$), with
211 increased ACH in the room more likely to produce higher C_T values, thus lower viral loads (Figure
212 5b). However, a significant decrease in the percent positivity of aerosol samples was not observed (P
213 $= 0.43$) as ACH increased across study rooms (Figure 5c).. Taken together, these results suggest that,
214 even across a fairly narrow and low range of ACH, increased ventilation rate decreases the
215 detectable aerosolized viral load within enclosed spaces. However, the lack of significance in the
216 decrease in percent positivity suggests that the modest range of ACH values found in this study is
217 not enough to decrease the abundance of viral particles in the enclosed space to an undetectable
218 level, thus suggesting higher ACH is required to support safer indoor congregation. Multiple articles
219 have previously hypothesized that increased ventilation rate would translate to lower airborne viral
220 loads^{22,67-70}. To our knowledge, this study demonstrates the first real-world experimental evidence of
221 increased ventilation within the built environment contributing to decreased aerosolized viral load.

222 One common method for increasing the ventilation that is available in the vast majority of BE's is
223 the operation of windows. Windows can dramatically increase the overall ACH within buildings and
224 other enclosed spaces⁷¹. In this case, opening a dorm room window will decrease the pressure on the
225 mechanical ventilation (the exhaust air fan in the bathroom) and increase the efficiency of air
226 movement by the exhaust fan⁷². More importantly, opening a window often increases the absolute
227 ACH (not just the measured ACH of the mechanical exhaust, in the room through increased air

228 movement in and out of the open window⁷³. In order to assess the potential impact of window
229 operations on the aerosolized viral load present within the study participant's rooms, study
230 participants were asked the status of their room windows during the course of the previous sampling
231 period and researchers observed current window operation status at each entry. Samples were split
232 into two groups consisting of (i) the window was open for more than 50% of the sampling period or
233 (ii) the window was open for less than 50% of the sampling period. Samples from aerosol collection
234 methods (AerosolSense and passive settling plates) demonstrated a significant increase in C_T values
235 (correlating with a decrease in viral load) when the window was open for more than 50% of the
236 sampling period (Figure 5d). These results suggest that the increased ventilation that is provided
237 from an open window has the ability to reduce the detectable viral load in the room by half (or
238 more) when windows are open ($\bar{x}=34.4$) compared to when the windows are closed ($\bar{x}=33.2$).
239 Window opening, as suggested by a variety of previous analyses and reviews^{69,74-78}, appears to
240 provide significant reduction in viral load while being a low-cost and low-labor intervention when
241 thermal control, security, and outdoor contaminants are not a concern.



242

243 **Figure 5. Impact of differential ventilation rates on SARS-CoV-2 RNA identification. (a)** Distribution of the
 244 calculated air exchanges per hour (ACH) from mechanical exhaust across all isolation rooms occupied by
 245 study participants. **(b)** Relationship between the observed cycle threshold (C_T) values and the air changes per
 246 hour (ACH) from occupied isolation rooms. The black line indicates fit from a linear model to the raw data
 247 and the grey area represents the 95% confidence interval for that model. Individual points are colored based on
 248 the ACH observed in that sample with darker colors representing lower ACH values and lighter colors
 249 representing higher ACH values. **(c)** Relationship between the observed percent positivity from each entry into
 250 a subject room and the air changes per hour (ACH) from occupied isolation rooms. The black line indicates fit
 251 from a linear model to the raw data and the grey area represents the 95% confidence interval for that model.
 252 Individual points are colored based on the ACH observed in that sample with darker colors representing lower
 253 ACH values and lighter colors representing higher ACH values. **(d)** Boxplots of observed cycle threshold (C_T)
 254 values of aerosol samples taken during periods when the window was open for more than 50% of the sampling
 255 period (yellow) or closed for more than 50% of the sampling period (purple), as recorded during the entry
 256 surveys answered by participants.

257 There are multiple limitations to note in our investigation. Our study population, made up of
 258 students living in the university residence halls, is inherently not a representative sample of the broad
 259 spectrum of individuals that may contract COVID-19. Particularly, our study population is
 260 composed of individuals between the ages of 18 and 24. The age of the individual suffering from

261 COVID-19 has been associated with altered levels of detectable SARS-CoV-2 RNA⁷⁹ and viral
262 shedding dynamics may differ from that seen in our investigation. Furthermore, our symptom and
263 window position results are largely based upon the results of self-reported survey data. This survey
264 data may suffer from inconsistencies and misclassification bias, particularly data pertaining to
265 symptom presence and severity⁸⁰⁻⁸³. Lastly, there is a lack of data demonstrating a presence or absence
266 of SARS-CoV-2 viability throughout the course of the study participants' time in the isolation
267 rooms. SARS-CoV-2 RNA has been demonstrated to remain within patient and environmental
268 samples, even when SARS-CoV-2 viability and infectiousness has ceased⁸⁴⁻⁸⁷.

269
270 Overall, we present a detailed longitudinal analysis of oral, nasal, and environmental viral loads
271 associated with individuals in a quarantine environment. We find that subject samples demonstrate a
272 decrease, but not a ceasing, in overall viral load as their quarantine period progresses. Based upon
273 the self-reported symptoms of study participants, we find that coughing and GI symptoms most
274 strongly correlate with increased environmental contamination, likely through an increase in virus
275 shedding during coughing and bowel activity and movements. Additionally, we demonstrate
276 significant differences in environmental contamination between symptomatic and asymptomatic
277 individuals, as well as between periods of intermittent positive and negative human samples. Lastly,
278 we provide the first real-world experiential evidence for decreased aerosol viral load with increasing
279 mechanical ventilation levels and demonstrate significantly reduced detectable SARS-CoV-2 in study
280 rooms with open windows compared with those with closed windows. These results are directly
281 applicable to those occupying common spaces with an individual known to be positive for COVID-
282 19. We demonstrate that even asymptomatic infection with SARS-CoV-2 can yield high levels of
283 environmental contamination. However, we also identified that increasing the total ACH within the
284 space occupied by the COVID-19 positive individual can aid in the reduction of the overall viral

285 load present in that environment. Furthermore, we add to the mounting evidence that SARS-CoV-2
286 is emitted by COVID-19 positive individuals which then disperse into the surrounding space as
287 potentially infectious aerosols which can be monitored through environmental surveillance
288 programs to support awareness and safety. We observe that indoor bioaerosols can be consistently
289 measured with a high-flow bioaerosol sampler and demonstrate utility in biosurveillance and to
290 assess mitigation effectiveness. Ideally, individuals would physically distance themselves from and
291 avoid shared air spaces with a COVID-19 positive individual, ensure the positive individual wears a
292 mask to reduce the quantity of emitted virus, and wear a mask themselves indoors. Ideally, building
293 operators would monitor indoor air for pathogenic bioaerosols and make preparations to increase
294 ventilation when pathogenic bioaerosols are present.

295

296 **Materials and Methods**

297 *Institutional Approval and Data Availability*

298 All protocols regarding to the handling of biological materials were reviewed and approved by
299 Advarra Institutional Biosafety Committee (IBC) (Protocol #PROTO202000132). Advarra IBC is
300 an authorized external IBC for the University of Oregon and is registered with the National Institute
301 of Health (NIH). All protocols relating to human subjects involved in the study were reviewed and
302 approved by the University of Oregon Institutional Review Board (IRB) (Protocol #12292020).

303 *Subject Recruitment*

304 University of Oregon COVID-19 protocols require individuals living in the residence halls to move
305 out of their current residence and occupy an isolation dormitory room during the course of their
306 isolation period (14 days). Individuals positive for COVID-19 were identified through the University
307 of Oregon Monitoring and Assessment Program (MAP)⁸⁸. Following transfer to the isolation

308 dormitory, individuals were recruited into the program for the duration of their stay at the isolation
309 dormitory or until they wished to be removed from the study.

310 *Subject Questionnaire*

311 During the first sampling period, study subjects verbally filled out a questionnaire (1st entry
312 questionnaire) that asked participants about their infection timeline, positive test date, age, biological
313 sex, race and ethnicity, recent travel history, lifestyle, medications taken, and symptom onset and
314 severity. Additionally, study subjects verbally completed a followup questionnaire during each
315 subsequent entry into the room to track their symptoms, medications taken, and the status of the
316 study room windows. The symptoms that were tracked included fever, coughing, sneezing, difficulty
317 breathing, fatigue, headache, aching eyes, watering eyes, sore throat, distorted taste, loss of taste,
318 distorted smell, loss of smell, ringing ears, gastrointestinal (GI) symptoms, congestion, and brain fog.
319 Study participants indicated whether or not they were currently experiencing any of the surveyed or
320 other symptoms and the severity on a scale of 1-5, with 5 being the most severe. All survey answers
321 were self-reported by the study participants.

322 *Airflow Monitoring*

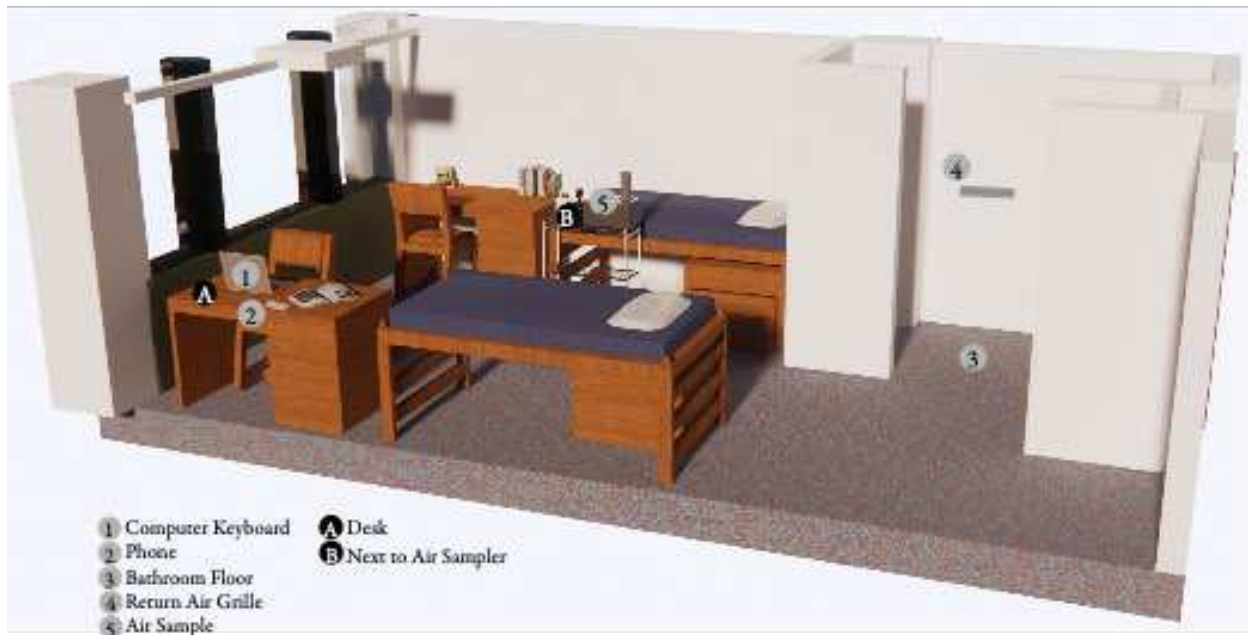
323 The rate of air exhausted from the isolation rooms were determined for each room. The only
324 location which is designed to exhaust air from the rooms is through the exhaust air vent located in
325 the bathroom of each unit or an open window. The room air is supplied from either the building
326 common areas (via a roof-top unit supplying 100% outside air) or the dormitory room windows.
327 The velocity of exhausted air from each room was measured by placing a customized adapter with a
328 three inch diameter outlet that rested against the exhaust air grille structural perimeter. A hot wire
329 anemometer (TSI Incorporated, model #9565) with probe (TSI Incorporated, model #964)
330 measured the velocity of flow at the center. The measurement was converted to volumetric flow rate

331 using the equation $VF = \frac{0.9 * \pi * 0.25^2}{4} * V$, where V is the measured velocity at the center in feet per
332 minute, 0.25 is the three inch diameter outlet converted to feet, and 0.9 is the conversion factor
333 accounting for peak flow at the center and averaging flow across the area of the hole. The air
334 changes per hour (ACH) flow rate was calculated using the dimensions of the study rooms as
335 described in the architectural plans and the equation $ACH_F = \frac{VF * 60}{v}$, where v is the volume of the
336 room in cubic feet, 60 is the minutes in an hour, and VF is the calculated volumetric flow rate.
337 Measurements were taken with (1) the hall door, exterior window and, bathroom door closed, and
338 (2) the hall door closed and the exterior window and bathroom door open.

339 *Sample Collection*

340 Samples were collected 3-5 times throughout a day with approximately two hours lapsing between
341 subsequent sampling times. At each entry, both a mouth and shallow nasal swab were collected from
342 the study participant. Environmental samples were collected through environmental swabs, passive
343 air settling plates, and active air sampling (Figure 6). Environmental swabs were collected from the
344 participant's cell phone, computer, bathroom floor, and exhaust air grille located within the
345 bathroom. Flocked nylon fiber oropharyngeal swabs (Typenex Medical LLC, Catalog #SW0202)
346 pre-moistened with DNA/RNA Shield (Zymo Research, Catalog #R1100) were used to thoroughly
347 swab the sampling location (sampling area $\sim 600 \text{ cm}^2$, except for smaller items such as cellphones)
348 for 15-20 seconds and returned to 1 mL of DNA/RNA Shield. Subject phones and computers were
349 cleaned with bleach wipes following sampling to remove the residue left behind by the DNA/RNA
350 shield. Settled particulates were captured using both components (base and lid) of standard Petri
351 dishes (Corning Scientific). Following the sampling period, both sides of the Petri dish (sampling
352 area $\sim 110 \text{ cm}^2$) were swabbed following the protocol described above for environmental swabs.
353 Active air samples were collected using the AerosolSense 2900 sampler (Thermo Scientific, Catalog

354 #121561-00). The AerosolSense sampler works by drawing air into an accelerating impactor at a rate
355 of 200 L/minute, causing particles to impact onto a collection substrate. Following the sampling
356 period, the collection substrate was transferred to 1 mL of DNA/RNA Shield using sterilized
357 forceps and transported back to the laboratory. Upon return to the laboratory, the capture media
358 was briefly vortexed, then centrifuged for two minutes at 1,500 x g to collect all liquid from the
359 collection substrate. Following centrifugation, the collection substrate was discarded.



360
361 **Figure 6. Representative layout of study rooms and sampling locations. Numbers in grey circles represent**
362 **locations sampled with flocked swabs and letters in black circles represent locations sampled through passive**
363 **air settling plates. Sampling location 5 represents the active air sample collected with the AerosolSense**
364 **Sampler**

365 *Molecular Analysis*

366 All protocols were performed in a Purifier Logic+ Class II, Type A2 biosafety cabinet (LabConco,
367 Catalog #302420001). An aliquot of 400 μ L of each sample was used as the input for RNA
368 extraction using the Quick-DNA/RNA Viral Magbead kit (Zymo Research, Catalog #R2141)
369 following the manufacturer's protocol. Briefly, 800 μ L of lysis buffer and 20 μ L magnetic beads
370 were added to each well, the plate was sealed, and shaken continuously for 10 minutes. Following
371 the ten minute incubation, the supernatant was removed, and the lysates were washed four times

372 (1X with MagBead DNA/RNA Wash 1, 1X MagBead with DNA/RNA Wash 2, 2X with 100%
373 ethanol). Nucleic acids were eluted into 50 μ L nuclease-free water and stored at -80°C until
374 downstream analysis. Successful RNA extraction was confirmed in each sample through the addition
375 of a 5 μ L spike-in of Escherichia coli MS2 bacteriophage into each extraction well. Each extraction
376 plate also contained one extraction control containing nuclease-free water instead of sample.

377 All samples underwent quantitative reverse-transcription polymerase chain reaction (qRT-PCR)
378 analysis using the TaqPath COVID-19 Combo Kit (Thermo Fisher Scientific, Catalog #A47814).
379 This quadruplex qRT-PCR reaction targets the spike (S), nucleocapsid (N), and RNA-dependent
380 RNA polymerase (RdRP/ORF1ab) genomic regions. Additionally, the assay also targets the
381 Escherichia coli MS2 bacteriophage as an internal process control. The reaction mixtures included 5
382 μ L TaqPath 1-Step Multiplex Mastermix without ROX (Thermo Fisher Scientific, Catalog
383 #A28521), 9 μ L nuclease-free water (Invitrogen, Catalog #4387936), 1 μ L COVID-19 Real Time
384 PCR Assay Multiplex Mix (Thermo Fisher Scientific, Catalog #A47814), and 5 μ L of template RNA.
385 Thermocycling was performed with the QuantStudio5 (Applied Biosystems) using the following
386 cycling conditions: 25°C for 2 minutes, 53°C for 10 minutes, 95°C for 2 minutes, and 40 cycles of
387 95°C for 3 seconds and 60°C for 30 seconds. Samples were considered positive if amplification was
388 observed in two of three genome targets with a cycle threshold (CT) value less than or equal to 35
389 ($C_T < 35$)⁸⁹. Each qRT-PCR plate contained a positive RNA control, a no-template control (nuclease-
390 free water), and three extraction controls. All controls performed as expected.

391 *Statistical Analyses*

392 Analyses were performed using the statistical programming environment R⁹⁰. Associations between
393 observed C_T values and study subject symptoms were identified through the use of a generalized
394 linear model of the form $y = \beta_1(x_1) + \beta_2(x_2) + \dots + \beta_n(x_n) + E$ where y is the observed C_T , β_i

395 values are linear regression coefficients for fixed effects x_i , and E is a vector of errors. Significant
396 changes in C_T values over time were identified through linear mixed models of the form $y_i = X_i\beta +$
397 $Z_iu_i + \epsilon_i$ ^{91,92} using a restricted maximum likelihood (REML) approach and including the individual
398 occupying the room as a random effect. Student's t-tests were used to compare differences in
399 observed C_T values between sampling groups. Differences were considered significant with $P < 0.05$.

400 **Data and Code Availability**

401 All data and code supporting this study and required to recreate the analyses are deposited in Github
402 at <https://github.com/BioBE/UO-COVID-Dorms>.

403 **References**

- 404 1. National Academies of Sciences, Engineering, and Medicine *et al. Microbiomes of the built*
405 *environment: A research agenda for indoor microbiology, human health, and buildings*. (National
406 Academies Press, 2017).
- 407 2. Horve, P. F. *et al.* Building upon current knowledge and techniques of indoor microbiology
408 to construct the next era of theory into microorganisms, health, and the built environment. *J.*
409 *Expo. Sci. Environ. Epidemiol.* 1–17 (2019).
- 410 3. Organization, W. H. & Others. World health organization coronavirus disease (COVID-19)
411 dashboard. (2020).
- 412 4. Lu, J. *et al.* COVID-19 outbreak associated with air conditioning in restaurant, guangzhou,
413 china, 2020. *Emerging Infectious Diseases* **26**, 1628–1631 (2020).
- 414 5. Lednicky, J. A. *et al.* Collection of SARS-CoV-2 virus from the air of a clinic within a
415 university student health care center and analyses of the viral genomic sequence. *Aerosol and*
416 *Air Quality Research* vol. 20 1167–1171 (2020).
- 417 6. Lednicky, J. A. *et al.* Viable SARS-CoV-2 in the air of a hospital room with COVID-19
418 patients. *medRxiv* (2020).
- 419 7. Hamner, L. *et al.* High SARS-CoV-2 attack rate following exposure at a choir practice skagit
420 county, washington, march 2020. *MMWR. Morbidity and Mortality Weekly Report* **69**, 606–610
421 (2020).
- 422 8. Razzini, K. *et al.* SARS-CoV-2 RNA detection in the air and on surfaces in the COVID-19
423 ward of a hospital in milan, italy. *Sci. Total Environ.* **742**, 140540 (2020).

- 424 9. Morawska, L. & Cao, J. Airborne transmission of SARS-CoV-2: The world should face the
425 reality. *Environ. Int.* **139**, 105730 (2020).
- 426 10. Allen, J. G. & Marr, L. C. Recognizing and controlling airborne transmission of SARS-CoV-
427 2 in indoor environments. *Indoor Air* vol. 30 557–558 (2020).
- 428 11. Liu, J., Huang, J. & Xiang, D. Large SARS-CoV-2 outbreak caused by asymptomatic traveler,
429 china. *Emerging Infectious Diseases* **26**, 2260–2263 (2020).
- 430 12. Speake, H. *et al.* Flight-associated transmission of severe acute respiratory syndrome
431 coronavirus 2 corroborated by whole-genome sequencing. *Emerging Infectious Diseases* **26**,
432 2872–2880 (2020).
- 433 13. Katelaris, A. L. *et al.* Epidemiologic evidence for airborne transmission of SARS-CoV-2
434 during church singing, australia, 2020. *Emerging Infectious Diseases* **27**, 1677–1680 (2021).
- 435 14. Nissen, K. *et al.* Long-distance airborne dispersal of SARS-CoV-2 in COVID-19 wards. *Sci.*
436 *Rep.* **10**, 19589 (2020).
- 437 15. Somsen, G. A., Rijn, C. van, Kooij, S., Bem, R. A. & Bonn, D. Small droplet aerosols in
438 poorly ventilated spaces and SARS-CoV-2 transmission. *Lancet Respir Med* **8**, 658–659 (2020).
- 439 16. Dumont-Leblond, N. *et al.* Positive no-touch surfaces and undetectable SARS-CoV-2
440 aerosols in long-term care facilities: An attempt to understand the contributing factors and
441 the importance of timing in air sampling campaigns. *Am. J. Infect. Control* (2021).
- 442 17. Correia, G., Rodrigues, L., Gameiro da Silva, M. & Gonçalves, T. Airborne route and bad
443 use of ventilation systems as non-negligible factors in SARS-CoV-2 transmission. *Med.*
444 *Hypotheses* **141**, 109781 (2020).
- 445 18. Ye, G. *et al.* Environmental contamination of SARS-CoV-2 in healthcare premises. *J. Infect.*
446 (2020).
- 447 19. Ryu, B.-H. *et al.* Environmental contamination of SARS-CoV-2 during the COVID-19
448 outbreak in south korea. *Am. J. Infect. Control* **48**, 875–879 (2020).
- 449 20. Ahn, J. Y. *et al.* Environmental contamination in the isolation rooms of COVID-19 patients
450 with severe pneumonia requiring mechanical ventilation or high-flow oxygen therapy. *Journal*
451 *of Hospital Infection* vol. 106 570–576 (2020).
- 452 21. Jin, T. *et al.* SARS-CoV-2 presented in the air of an intensive care unit (ICU). *Sustain Cities Soc*
453 **65**, 102446 (2021).
- 454 22. Cheng, V. C.-C. *et al.* Air and environmental sampling for SARS-CoV-2 around hospitalized
455 patients with coronavirus disease 2019 (COVID-19). *Infection Control & Hospital Epidemiology*
456 **41**, 1258–1265 (2020).
- 457 23. Fernández-de-Mera, I. G. *et al.* Detection of environmental SARS-CoV-2 RNA in a high
458 prevalence setting in spain. *Transbound. Emerg. Dis.* **68**, 1487–1492 (2021).

- 459 24. Li, Y. H., Fan, Y. Z., Jiang, L. & Wang, H. B. Aerosol and environmental surface monitoring
460 for SARS-CoV-2 RNA in a designated hospital for severe COVID-19 patients. *Epidemiol.*
461 *Infect.* **148**, e154 (2020).
- 462 25. Escudero, D. *et al.* SARS-CoV-2 analysis on environmental surfaces collected in an intensive
463 care unit: Keeping Ernest Shackleton's spirit. *Intensive Care Med Exp* **8**, 68 (2020).
- 464 26. Environmental surface and air sampling in the context of the COVID-19 pandemic.
- 465 27. Ong, S. W. X. *et al.* Air, surface environmental, and personal protective equipment
466 contamination by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) from a
467 symptomatic patient. *JAMA* (2020).
- 468 28. Coil, D. A. *et al.* SARS-CoV-2 detection and genomic sequencing from hospital surface
469 samples collected at UC Davis. *medRxiv* (2021).
- 470 29. Coil, D. A. *et al.* SARS-CoV-2 detection and genomic sequencing from hospital surface
471 samples collected at UC Davis. *PLOS ONE* **16**, e0253578 (2021).
- 472 30. Borges, J. T., Nakada, L. Y. K., Maniero, M. G. & Guimarães, J. R. SARS-CoV-2: A
473 systematic review of indoor air sampling for virus detection. *Environ. Sci. Pollut. Res. Int.*
474 (2021).
- 475 31. Horve, P. F. *et al.* Identification of SARS-CoV-2 RNA in healthcare heating, ventilation, and
476 air conditioning units. *Infectious Diseases (except HIV/AIDS)* (2020).
- 477 32. Hermes, A. C. *et al.* Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2)
478 environmental contamination and childbirth. *Obstet. Gynecol.* **136**, 827–829 (2020).
- 479 33. Doremalen, N. van *et al.* Aerosol and surface stability of SARS-CoV-2 as compared with
480 SARS-CoV-1. *N. Engl. J. Med.* (2020).
- 481 34. Lednicky, J. A. *et al.* Viable SARS-CoV-2 in the air of a hospital room with COVID-19
482 patients. *Int. J. Infect. Dis.* **100**, 476–482 (2020).
- 483 35. Joukar, F. *et al.* Persistence of SARS-CoV-2 RNA in the nasopharyngeal, blood, urine, and
484 stool samples of patients with COVID-19: a hospital-based longitudinal study. *Virology*
485 *Journal* **18**, (2021).
- 486 36. Aranha, C., Patel, V., Bhor, V. & Gogoi, D. Cycle threshold values in RT-PCR to determine
487 dynamics of SARS-CoV-2 viral load: An approach to reduce the isolation period for
488 COVID-19 patients. *Journal of Medical Virology* (2021) doi:10.1002/jmv.27206.
- 489 37. Coil, D. A. *et al.* SARS-CoV-2 detection and genomic sequencing from hospital surface
490 samples collected at UC Davis. *PLOS ONE* **16**, e0253578 (2021).
- 491 38. Zhou, Y., Zeng, Y. & Chen, C. Presence of SARS-CoV-2 RNA in isolation ward
492 environment 28 days after exposure. *International Journal of Infectious Diseases* **97**, 258–259
493 (2020).

- 494 39. Lavezzo, E. *et al.* Suppression of a SARS-CoV-2 outbreak in the Italian municipality of Vo'.
495 *Nature* **584**, 425–429 (2020).
- 496 40. Finelli, C. & Parisi, S. The clinical impact of COVID-19 epidemic in the hematologic setting.
497 *Advances in Biological Regulation* **77**, 100742 (2020).
- 498 41. Arons, M. M. *et al.* Presymptomatic SARS-CoV-2 Infections and Transmission in a Skilled
499 Nursing Facility. *New England Journal of Medicine* **382**, 2081–2090 (2020).
- 500 42. Zhou, R. *et al.* Viral dynamics in asymptomatic patients with COVID-19. *International Journal*
501 *of Infectious Diseases* **96**, 288–290 (2020).
- 502 43. Wei, W. E. *et al.* Presymptomatic transmission of SARS-CoV-2 singapore, january 23march
503 16, 2020. *MMWR. Morbidity and Mortality Weekly Report* **69**, 411–415 (2020).
- 504 44. Bernal, J. L. *et al.* Transmission dynamics of COVID-19 in household and community
505 settings in the united kingdom. (2020).
- 506 45. Decker, A. *et al.* Prolonged SARS-CoV-2 shedding and mild course of COVID-19 in a
507 patient after recent heart transplantation. *American Journal of Transplantation* **20**, 3239–3245
508 (2020).
- 509 46. Folgueira, M. D., Luczkowiak, J., Lasala, F., Pérez-Rivilla, A. & Delgado, R. Persistent
510 SARS-CoV-2 replication in severe COVID-19. (2020).
- 511 47. van Kampen, J. J. A. *et al.* Shedding of infectious virus in hospitalized patients with
512 coronavirus disease-2019 (COVID-19): Duration and key determinants. (2020).
- 513 48. Riediker, M. & Tsai, D.-H. Estimation of Viral Aerosol Emissions From Simulated
514 Individuals With Asymptomatic to Moderate Coronavirus Disease 2019. *JAMA Network*
515 *Open* **3**, e2013807 (2020).
- 516 49. Wang, Y., Xu, G. & Huang, Y.-W. Modeling the load of SARS-CoV-2 virus in human
517 expelled particles during coughing and speaking. *PLOS ONE* **15**, e0241539 (2020).
- 518 50. Riediker, M. & Tsai, D.-H. Estimation of Viral Aerosol Emissions From Simulated
519 Individuals With Asymptomatic to Moderate Coronavirus Disease 2019. *JAMA Network*
520 *Open* **3**, e2013807 (2020).
- 521 51. Peccia, J. *et al.* Measurement of SARS-CoV-2 RNA in wastewater tracks community
522 infection dynamics. *Nature Biotechnology* **38**, 1164–1167 (2020).
- 523 52. Parasa, S. *et al.* Prevalence of Gastrointestinal Symptoms and Fecal Viral Shedding in
524 Patients With Coronavirus Disease 2019. *JAMA Network Open* **3**, e2011335 (2020).
- 525 53. V'kovski, P. *et al.* Disparate temperature-dependent virus-host dynamics for SARS-CoV-2
526 and SARS-CoV in the human respiratory epithelium. *PLOS Biology* **19**, e3001158 (2021).
- 527 54. Aljishi, J. M. & Al-Tawfiq, J. A. Intermittent viral shedding in respiratory samples of patients
528 with SARS-CoV-2: observational analysis with infection control implications. *Journal of*
529 *Hospital Infection* **107**, 98–100 (2021).

- 530 55. Li, N., Wang, X. & Lv, T. Prolonged SARS-CoV-2 RNA shedding: Not a rare phenomenon.
531 *Journal of Medical Virology* **92**, 2286–2287 (2020).
- 532 56. Liu, W.-D. *et al.* Prolonged virus shedding even after seroconversion in a patient with
533 COVID-19. *Journal of Infection* **81**, 318–356 (2020).
- 534 57. Lau, M. S. Y. *et al.* Characterizing superspreading events and age-specific infectiousness of
535 SARS-CoV-2 transmission in Georgia, USA. *Proceedings of the National Academy of Sciences* **117**,
536 22430–22435 (2020).
- 537 58. Parhizkar, H., Van Den Wymelenberg, K., Haas, C. & Corsi, R. A quantitative risk
538 estimation platform for indoor aerosol transmission of COVID-19. (2021).
- 539 59. Majra, D., Benson, J., Pitts, J. & Stebbing, J. SARS-CoV-2 (COVID-19) superspreader
540 events. *J. Infect.* **82**, 36–40 (2021).
- 541 60. Hamner, L. High SARS-CoV-2 attack rate following exposure at a choir practice—skagit
542 county, washington, march 2020. *MMWR Morb. Mortal. Wkly. Rep.* **69**, (2020).
- 543 61. Jianyun Lu *et al.* COVID-19 outbreak associated with air conditioning in restaurant,
544 guangzhou, china, 2020. *Emerging Infectious Disease journal* **26**, (2020).
- 545 62. Speake, H. *et al.* Flight-associated transmission of severe acute respiratory syndrome
546 coronavirus 2 corroborated by whole-genome sequencing. *Emerging Infectious Diseases* **26**,
547 2872–2880 (2020).
- 548 63. Katelaris, A. L. *et al.* Epidemiologic evidence for airborne transmission of SARS-CoV-2
549 during church singing, australia, 2020. *Emerging Infectious Diseases* **27**, 1677–1680 (2021).
- 550 64. Thanh, H. N. *et al.* Outbreak investigation for COVID-19 in northern Vietnam. *The Lancet*
551 *Infectious Diseases* **20**, 535–536 (2020).
- 552 65. Escandón, K. *et al.* COVID-19 false dichotomies and a comprehensive review of the
553 evidence regarding public health, COVID-19 symptomatology, SARS-CoV-2 transmission,
554 mask wearing, and reinfection. *BMC Infectious Diseases* **21**, (2021).
- 555 66. The American Society of Heating, Refrigerating and Air Condition Engineers, Inc.
556 (ASHRAE). *Ventilation of health care facilities (ANSI/ASHRAE/ASHE standard 170-2017)*.
557 (2017).
- 558 67. Allen, J. G. & Ibrahim, A. M. Indoor Air Changes and Potential Implications for SARS-
559 CoV-2 Transmission. *JAMA* **325**, 2112 (2021).
- 560 68. Chia, P. Y. *et al.* Detection of air and surface contamination by SARS-CoV-2 in hospital
561 rooms of infected patients. *Nature Communications* **11**, (2020).
- 562 69. Dietz, L. *et al.* 2019 Novel Coronavirus (COVID-19) Pandemic: Built Environment
563 Considerations To Reduce Transmission. *mSystems* **5**, (2020).
- 564 70. Klompas, M., Baker, M. A. & Rhee, C. Airborne Transmission of SARS-CoV-2. *JAMA* **324**,
565 441 (2020).

- 566 71. Howard-Reed, C., Wallace, L. A. & Ott, W. R. The Effect of Opening Windows on Air
567 Change Rates in Two Homes. *Journal of the Air & Waste Management Association* **52**, 147–159
568 (2002).
- 569 72. Qian, H. *et al.* Natural ventilation for reducing airborne infection in hospitals. *Building and*
570 *Environment* **45**, 559–565 (2010).
- 571 73. Escombe, A. R. *et al.* Natural Ventilation for the Prevention of Airborne Contagion. *PLoS*
572 *Medicine* **4**, e68 (2007).
- 573 74. Morawska, L. *et al.* How can airborne transmission of COVID-19 indoors be minimised?
574 *Environment International* **142**, 105832 (2020).
- 575 75. Nembhard, M. D., Burton, D. J. & Cohen, J. M. Ventilation use in nonmedical settings
576 during COVID-19: Cleaning protocol, maintenance, and recommendations. *Toxicology and*
577 *Industrial Health* **36**, 644–653 (2020).
- 578 76. Mathai, V., Das, A., Bailey, J. A. & Breuer, K. Airflows inside passenger cars and
579 implications for airborne disease transmission. *Science Advances* **7**, eabe0166 (2020).
- 580 77. Bhagat, R. K., Davies Wykes, M. S., Dalziel, S. B. & Linden, P. F. Effects of ventilation on
581 the indoor spread of COVID-19. *Journal of Fluid Mechanics* **903**, (2020).
- 582 78. Jarvis, M. C. Aerosol transmission of SARS-CoV-2: Physical principles and implications.
583 *Frontiers in Public Health* **8**, (2020).
- 584 79. Zhou, C. *et al.* Impact of age on duration of viral RNA shedding in patients with COVID-19.
585 *Aging* (2020) doi:10.18632/aging.104114.
- 586 80. Daniali, H. & Flaten, M. A. What psychological factors make individuals believe they are
587 infected by coronavirus 2019? *Frontiers in Psychology* **12**, (2021).
- 588 81. Kluytmans-van den Bergh, M. F. Q. *et al.* Prevalence and Clinical Presentation of Health
589 Care Workers With Symptoms of Coronavirus Disease 2019 in 2 Dutch Hospitals During an
590 Early Phase of the Pandemic. *JAMA Network Open* **3**, e209673 (2020).
- 591 82. Nomura, S. *et al.* An assessment of self-reported COVID-19 related symptoms of 227,898
592 users of a social networking service in Japan: Has the regional risk changed after the
593 declaration of the state of emergency? *The Lancet Regional Health - Western Pacific* **1**, 100011
594 (2020).
- 595 83. Merckelbach, H., Dandachi-FitzGerald, B., van Helvoort, D., Jelicic, M. & Otgaar, H. When
596 Patients Overreport Symptoms: More Than Just Malingering. *Current Directions in Psychological*
597 *Science* **28**, 321–326 (2019).
- 598 84. Zhang, Y. *et al.* Prevalence and Persistent Shedding of Fecal SARS-CoV-2 RNA in Patients
599 With COVID-19 Infection: A Systematic Review and Meta-analysis. *Clinical and Translational*
600 *Gastroenterology* **12**, e00343 (2021).
- 601 85. Rawlings, S. A. *et al.* No Evidence of SARS-CoV-2 Seminal Shedding Despite SARS-CoV-2
602 Persistence in the Upper Respiratory Tract. *Open Forum Infectious Diseases* **7**, (2020).

- 603 86. Yang, J.-R. *et al.* Persistent viral RNA positivity during the recovery period of a patient with
604 SARS-CoV-2 infection. *Journal of Medical Virology* **92**, 1681–1683 (2020).
- 605 87. Kang, H., Wang, Y., Tong, Z. & Liu, X. Retest positive for SARS-CoV-2 RNA of
606 “recovered” patients with COVID-19: Persistence, sampling issues, or re-infection? *Journal of*
607 *Medical Virology* **92**, 2263–2265 (2020).
- 608 88. University of Oregon. Monitoring and assessment program (MAP).
- 609 89. TaqPath COVID-19 combo kit - instructions for use.
- 610 90. Team, R. C. & Others. R: A language and environment for statistical computing. (2013).
- 611 91. Laird, N. M. & Ware, J. H. Random-effects models for longitudinal data. *Biometrics* **38**, 963–
612 974 (1982).
- 613 92. Raudenbush, S. W. & Bryk, A. S. *Hierarchical linear models: Applications and data analysis methods*.
614 (SAGE, 2002).

615

616 **Acknowledgements**

617 The authors would like to thank Chuck Williams and Tony Schaffer, University of Oregon, for their
618 aid in forming a relationship with Thermo Fisher Scientific. The authors would like to thank
619 countless staff members from the University of Oregon. The authors would like to acknowledge the
620 tireless team at BioBE for all of their help, including Surbhi Nahata, Kate Qi, Jackson Mestler, Dan
621 Richards, Kathrine Tush, Liliya Zagorski, and Ellie Thacker. Most importantly, we would like to
622 extend a special thanks to the study participants who welcomed us during a very stressful event in
623 their lives.

624 **Author Contributions**

625 KGVDW performed funding acquisition and managed the investigation team. PFH, LGD, and
626 KGVDW conceived of project scope and methodology. KGVDW and LGD enrolled and
627 consented study participants. GB and PFH performed data curation and initial data exploration.
628 PFH developed final analysis scripts, performed final analysis, and created visualizations. PFH,

629 LGD, GB, GM, AOM, DN, VM, LB, and HP collected field samples and performed laboratory
630 analyses. PFH developed the original manuscript with direction and input from KGVDW. LGD,
631 GB, GM, AOM, DN, VM, LB, HP, and KGVDW provided manuscript revisions and edits on
632 subsequent manuscript drafts.

633 **Competing Interests Statement**

634 KGVDW has a company called Duktile through which he provides healthy building consulting,
635 including consulting related to viral pathogens, and he serves as a scientific advisor to EnviralTech, a
636 company that conducts viral surface surveillance, including in senior care facilities. No other authors
637 have any competing interests to declare.