

Supplemental Materials

A population framework for predicting the proportion of people infected by the far-field airborne transmission of SARS-CoV-2 indoors

Christopher Iddon^a, Benjamin Jones^{a,*}, Patrick Sharpe^a, Muge Cevik^b,
Shaun Fitzgerald^c

^a*Department of Architecture and Built Environment, University of Nottingham,
Nottingham, UK*

^b*Infection and Global Health Division, School of Medicine, University of St Andrews, St
Andrews, UK*

^c*Department of Engineering, Cambridge University, Cambridge, UK*

Keywords: relative exposure index, airborne, ventilation, aerosols,
transmission risk, viral load, COVID-19,

*Corresponding author

Email address: benjamin.jones@nottingham.ac.uk (Benjamin Jones)

Preprint submitted to Indoor Air

February 3, 2022

1. Model uncertainties

The output of the REI is a dose value of RNA copies that deposit onto the respiratory tract of a susceptible individual (for brevity we will term this the inhaled dose). The number of viral genome copies (RNA copies) is proportional to the number of viable virion, but the ratio of RNA copies to viable virion is unknown. Whether the deposited virion then leads to an infection in the susceptible individual depends upon the dose response – another unknown quantity, and the susceptibility of the individual to infection.

1.1. Uncertainty in dose response

There is currently no dose–response curve for SARS-CoV-2, however a number of studies have used a proposed dose curve for SARS-CoV-1, which is a typical coronavirus dose curve [1, 2, 3]. This dose curve was generated from inoculating four groups of transgenic mice (mice genetically modified to express the the human protein that is the receptor for the SARS-CoV-1 virus). The dose response curve was fitted to data from these four groups, in three of which all of the mice became infected and in one group a third of mice become infected. This is a limited data set for curve fitting, although it is sufficient to assume that the dose curve is exponential rather than a Beta-Poisson distribution. It should be noted that the dose response of humans may vary significantly from that of transgenic mice.

Dose curves are fitted to low PFU but there is usually limited data at very low doses. Therefore, the dose-response relationship is highly uncertain at low doses and infection probabilities. This is important because, when considering a large population, even very low probabilities of infection could

25 lead to a significant number of infected people – and it may well be an
26 overestimation if the dose-response curve at very low level of virus is not
27 representative.

28 In another study Schijven *et al.* determined a model that 1440 RNA copies
29 was required to lead to an infection, drawing assumptions from the proportion
30 of isolated cultured SARS-CoV-2 (ie not collected from patient swabs) needed
31 to infect a cell culture line to calculate a PFU and then deriving an infectious
32 dose from a dose curve for human coronavirus 229E of 1,440 RNA copies
33 [4]. Uncertainties are that SARS-CoV-2 isolate unlikely to be comparable
34 to SARS-CoV-2 collected from patient samples eg swabs. The response of
35 a cell culture is unlikely to be comparable to a respiratory tract, mucosal
36 membranes and innate immunity of a human. The dose curve for 229E may
37 also be different to that for SARS-CoV-2.

38 There are challenges and uncertainties in the assumptions used to gener-
39 ate infective dose-response curves for SARS-CoV-2 and as these uncertainties
40 are not easily measurable, there will be unknown uncertainties in calculating
41 the probability of infection using such assumptions.

42 *1.2. Uncertainty in viral load*

43 It has been well established that the viral load of an infector increases from
44 the date of infection and is highest just before or at the onset of symptoms,
45 and as the disease progresses the viral load begins to reduce (within the
46 first week of symptom onset) [5, 6]. Viral load at any stage of infection
47 also varies between individuals, which increases the uncertainty in this value
48 [7, 8, 9, 10, 11, 12]. Some studies use reported cycle threshold values from
49 real time reverse transcription quantitative polymerase chain reaction (RT

50 qPCR) nasopharyngeal (NP) swabs to infer the viral load in respiratory fluid,
51 however this method assumes a direct correlation between the swab viral load
52 and the respiratory fluid viral load [13, 14]. RT qPCR is semi-quantitative
53 in that the number of cycles required to provide a positive signal for SARS-
54 CoV-2 genome is proportional to the starting amount of viral genome in
55 the sample. The greater the number of amplification cycles required, the
56 lower the starting amount of viral genome. A calibrated standard curve
57 can then be used to estimate the starting amount of viral genomic material.
58 However, the standard curve varies between test assays and different RT
59 qPCR thermal cyclers. This method assumes a complete doubling of genetic
60 material at each cycle, and because of the logarithmic relationship, the errors
61 in calculating the starting genomic material for low cycle counts are orders
62 of magnitude higher than those with high cycle counts. Additionally, the
63 estimated concentration of genomic material per unit volume is related to
64 the amount of genomic material in the buffer solution used in the assay,
65 not necessarily the amount in the patients respiratory fluid if data is from
66 NP swabs. The amount of genomic material added to the buffer solution is
67 dependent on not only the viral load of the patient, but also the quality of
68 NP sample collection, which is highly variable. Therefore, it is not possible
69 to determine absolute values of the viral load in patient’s respiratory fluid
70 using this method, although it is indicative of a range of variability – much
71 of which is likely to be proportional to the viral load of the individual at
72 the time the sample was collected. While it is somewhat correlated, recent
73 data suggests that the viral load of NP swabs may not reflect the amount
74 of infectious material present [10]. However, it is important to note that

75 there are wide variations in the measured genomic material in NP swabs
76 and that viral load in respiratory fluid is likely to vary over several orders of
77 magnitude, although absolute values and proportions are not determinable
78 with current data.

79 The RT-qPCR process also only amplifies a small section of viral genome
80 and is representative of viral genomic material in the original sample. Some of
81 this genomic material will be fragments, and therefore quantities of genomic
82 material are not representative of the number of viable virions in the original
83 sample, although likely to be proportional to, and there is some evidence
84 in the literature to suggest there is some correlation between Ct values and
85 infectious virus [6]. Additionally, one study of the influenza virus showed
86 that the viral load in NP swabs was not a significant predictor of aerosol
87 shedding [15]. In other studies the SARS-CoV-2 viral load of saliva has been
88 estimated using qRT-PCR that also show wide variability of several orders
89 of magnitude, although it is unknown if the saliva viral load is the same as
90 the viral concentration in the fluid of the respiratory tract[16].

91 *1.2.1. Viral load in aerosols*

92 If the viral load in respiratory fluid could be determined it is currently
93 unclear whether the viral concentration in respiratory aerosols and droplets
94 is uniformly distributed. Some studies suggest that the amount of virion in
95 smaller aerosols ($< 1 \mu\text{m}$) is higher than would be expected given the viral
96 concentration in the respiratory fluid [17, 18] and that there may be more
97 genomic material in the smallest aerosols [19]. There is also high variability
98 in the total volume of aerosols generated per unit volume of exhaled breath
99 between individuals, which is especially true for breathing and is dependent

100 upon the respiratory activity and respiratory capacity (e.g. talking, singing)
101 [20, 21, 22, 23]. A recent study from Coleman *et al.* [19] has detected SARS-
102 CoV-2 genomic material in expired aerosols from *some* Covid patients,
103 although 41% percent of patients exhaled no detectable genomic material.
104 Singing and talking generally produced more genomic material than breath-
105 ing, but there was large variability between patients. This suggests that res-
106 piratory activities that have previously been shown to increase aerosol mass,
107 also increase the amount of viral genomic material, although in this study
108 the viral concentration in aerosols cannot be determined because the mass
109 of aerosols generated was not measured. It also shows that the variability in
110 the amount of genomic material measured in expired aerosols is consistent
111 with the variability of viral loads as measured by swabs and saliva [19]. Sim-
112 ilarly Adenaiye *et al.* have also detected genomic material in aerosols from
113 patients infected with SARS-CoV-2 providing a sampled of exhaled air with
114 some talking and singing. Genomic material was most likely to be detected in
115 exhaled aerosols when the viral load of saliva or Mid-turbinate swabs (MTS)
116 was high ($> 10^8 RNA\ copies$ and $> 10^6$ for MTS and saliva samples respec-
117 tively). Additionally they were also able to culture viable virus from $< 2\%$
118 of fine aerosol samples (although one culture positive sample was from a fine
119 aerosol sample which has a less than Limit Of Detection amount of genomic
120 material as measured by RT-PCR, so could be an artefact). Providing some
121 evidence to support the epidemiological evidence that viable virus can exist
122 in exhaled aerosols [24].

123 Buonanno *et al.*, although noting that there are no values available in the
124 literature, propose a method to convert viral load to quanta emission rate

125 (where a quantum is defined as the dose of airborne droplet nuclei required to
126 cause infection in 63% of susceptible people) using a value for PFU per quanta
127 derived from Watanabe *et al.*, see Section 1.1, and RNA copies per PFU from
128 Fear *et al.* - values derived from stock SARS-CoV-2 created from Vero E6
129 cells, values which may well not reflect the quanta emission rate in an infected
130 person [25, 1, 26]. There is likely much uncertainty in this method and how
131 representative it is of infector viral emission rates.

132 1.3. Estimating viral emission from viral load

133 Although we have a range of viral loads for infectors in RNA copies per
134 ml, estimating how that relates to the emission of rna per unit time is
135 challenging due to the uncertainties listed, however, if we assume that the
136 RNA copies concentration is constant in aerosols and NP swabs we can use
137 the assumptions of Jones *et al.* [27] to convert a NP viral load into a viral
138 shedding rate. This methodology is derived from the aerosol volume distri-
139 bution of different respiratory activities from Morawskwa *et al.* and is similar
140 to that used by Lelieveld *et al.* [28, 23]. Table 1 shows that for a viral load of
141 10^7 RNA copies per ml this would assume the RNA copies shedding per hour,
142 and for comparison median values from Coleman *et al.* study are given, in
143 which the measured collected RNA copies were from Covid patients with a
144 median Ct of 16 from the patient's diagnostic sample [19].

145

Table 1: Estimated RNA copies shedding rates from an infector with a viral load of 10^7 RNA copies per ml compared to measured RNA copies shedding rates from patients with a median Ct of 16 as measured by Coleman *et al.* *value calculated from breathing and talking values

	estimated	measured median
	$RNA\ copies\ h^{-1}$	$RNA\ copies\ h^{-1}$
Breathing	203	127
Voiced counting (talking)	967	1912
Vocalisation (singing)	6198	2856
Breathing:talking 25:75	394	573*

146 Additionally a recent pre-print from Adenaiye *et al.* has also measured
 147 viral genome in patients, infected with SARS-CoV-2 alpha variant, breath-
 148 ing with some talking in coarse ($5\mu m$) and fine ($\leq 5\mu m$) aerosols with a
 149 total geometric mean of 1440 $RNA\ copies\ h^{-1}$ (with a maximum of 3×10^5
 150 $RNA\ copies\ h^{-1}$) [24]. Although this is more than the estimated values in
 151 Table 1, the viral load as measured by genome copies from Mid-turbinate
 152 swabs (MTS) was generally orders of magnitude higher than 10^7 .

153 In the measured data we don't know the relationship between the PCR
 154 cycle threshold and the patient viral load in $RNA\ copies/ml$, however the cal-
 155 culated shedding rate of viral genome for a viral load of 10^7 RNA copies per ml
 156 is a reasonable fit to the Coleman *et al.* and Adenaiye *et al.* data.

157 Other studies have suggested that genome emission rates of patients could
 158 be of the order of $10^6\ RNA\ copies\ h^{-1}$. Miller *et al.* derived this value from
 159 RNA copies measured in small hospital rooms containing Covid patients (here
 160 the air sampling equipment is located quite close to the patient and some
 161 observation that patients face turned to face collector for some samples) [29,

162 14]. Whilst Ma *et al.* collected viral genome in exhaled breath of patients that
163 would suggest patients exhaled in the region of 7×10^4 to 7×10^6 RNA copies per
164 hour, though in this study the collection mechanism involved exhaling into a
165 small straw-like tube for 5 minutes, which could also become contaminated
166 with viral laden saliva, thus over estimating the viral load of the exhaled
167 breath [30]. Although these exhaled rates of viral genome are much greater
168 than those collected by Coleman *et al.*, Miller *et al.* notes that suggests that
169 around 1 : 1000 genome copies are likely to be infectious virion [31, 14].
170 Adenaiye *et al.* suggest that from MTS there is around 1 : 10^4 viable virus
171 per measured genome copies[24]. For this study we have made the assumption
172 that all genome copies are viable virion, which either over-estimates the likely
173 infectiousness if using the Coleman *et al.* data, or is similar to the Miller *et*
174 *al.* assumptions if the viable virion shedding rate is in the order of 1000
175 virion per hour.

176 For the proportion of persons infected analysis, the inhaled dose is calcu-
177 lated for all viral loads, it should be noted that the calculated RNA copies
178 shedding rate is assumed to scale linearly with viral load per ml of respi-
179 ratory fluids, such that a viral load of $10^8 \text{ RNA copies/ml}$ would have ten
180 fold greater RNA copies shedding rates per hour. For comparison, given
181 a viral shedding of 394 RNA copies per hour (assumed for a viral load of
182 $10^7 \text{ RNA copies/ml}$) would lead to an individual inhaled dose of around 2.2
183 and 0.2 RNA copies for the Small Office and Big Office scenarios respectively.

184 1.3.1. Comparison of viral emission from literature

185 Extrapolating data for viral shedding rates from the literature is challeng-
186 ing as often the estimated doses and the probability of infection do not align

187 with epidemiological evidence. Chen *et al.* suggests that the upper limit for
188 the total virion shedding rate for moderate talking is 6000 virions per hour.
189 This includes droplets up to $100\ \mu\text{m}$ and so we assume the evaporation and
190 suspension of all these droplets in the air, although it is unlikely. Given a
191 $600\ \text{m}^3$ 20 person office at $10\ \text{l s}^{-1}$ per person with an infected person shed-
192 ding at 100 virions per minute we would expect < 10 virions to deposit in the
193 respiratory tract of a susceptible person over an 8 hour day, which, from the
194 DeDiego *et al.* SARS-CoV-1 dose curve, is unlikely to lead to an infection.
195 This suggests that if the upper limit of viral shedding is unlikely to result
196 in an infection, than the more likely lower viral shedding rates will be even
197 more unlikely to give rise to infection [32, 12].

198 Using the shedding rate of 6000 virions per hour for the Skagit choir
199 (Miller *et al.* [14] suggest shedding at 1000 virions per hour would be a rea-
200 sonable estimate for this scenario) we expect a susceptible person to have
201 about 7 virions deposit in their respiratory tract over a 2.5 hour practice pe-
202 riod. Using the SARS-CoV-1 dose curve, this gives a probability of infection
203 of 0.02. Given that the secondary attack rate at the Skagit choir was over
204 85%, this would suggest that either the k value in the SARS-CoV-2 dose
205 curve (see Equation 2) is much smaller than that predicted for SARS-CoV-1
206 or these models have used assumptions that have under estimated the virion
207 shedding rate, even for the high viral emitter considered here.

208 Alternatively, the quanta metric could be used because it captures the
209 effects of virion shedding and the dose curve by associating secondary trans-
210 mission in a particular transmission event. The quanta for the Skagit event
211 is calculated by Miller *et al.*, where the dose is likely to be 1.19 ± 0.48 quanta.

212 Using the Wells-Riley model gives a probability of transmission of between
213 0.51 and 0.81. This suggests that the quanta method is a better fit in this
214 Skagit choir scenario, although this is to be expected because the quanta
215 emission rate is exclusively derived from the number of secondary transmis-
216 sion events that occurred during the scenario.

217 If we use the Skagit quanta emission rate in another scenario, say a UK
218 junior school classroom described by Jones *et al.* [27], then it is possible to
219 conclude the following: the emission rate for singing is $970 \pm 390 \text{ q h}^{-1}$ but
220 assume a 30-fold reduction for aerosol emission when breathing and assume
221 a child breath rate (q_{sus}) of $0.44 \text{ m}^3 \text{ h}^{-1}$, then the dose over a 7 hour exposure
222 period is 0.77 ± 0.31 quanta, giving a probability of infection between 0.37 and
223 0.66. Although transmission events do occur in school classrooms, there isn't
224 evidence to suggest such high rates of secondary far-field transmission occur
225 regularly. Secondary attack rates (for all routes of transmission) amongst
226 primary pupils have been recorded at less than 1% [33] (although more recent
227 observations on infection rates amongst UK school age children in Autumn
228 2021 suggests secondary attack rates are likely to be higher than this for the
229 Delta variant, however, still not at probability of infection between 0.37 and
230 0.66 [34]). This suggests that the quanta emission rate (as estimated in the
231 Skagit Choir scenario) is either extremely unlikely or it scenario-specific and
232 so it is inappropriate to use a quanta emission rate determined from a single
233 scenario and apply to another. Sze *et al.* also covers uncertainties in the use
234 of quanta models [35].

235 **2. Results and discussion: Effect of varying model assumptions on**
 236 ***PPI***

237 In addition to the results reported in the main paper, here we report
 238 the effect of various model assumptions on the *PPI* and *TR*. All standard
 239 scenario inputs are given in Tables 2 and 3.

Table 2: Scenario inputs and calculations of individual risk.

	Big Office Reference	Small Office Comparator
Number of occupants, N	50	5
Space Volume, V (m^3)	1500	150
<i>Per capita</i> volume, $V N^{-1}$ (m^3 per person)		30
Air flow rate, ψV (ls^{-1})	500	50
Air change rate, ψ (h^{-1})		1.2
Removal rate, ϕ (h^{-1})		2.26
Equivalent ventilation rate, ϕV (ls^{-1})	942	94.2
Exposure time, T (h)		8
Dose constant, k [32]		410
Respiratory tract absorption fraction, K		0.55
Viable fraction, v (%)		100
Viral load (RNA copies per ml) [36]		10^7
Respiratory activity, <i>breathing: talking</i> (%)		72:25
Volumetric ratio of exhaled droplets to exhaled air, V_{drop}^*		5.05×10^{-13}
Respiratory fluid density (ml m^{-3})		1.25^8
Respiratory rate, q_{resp} ($\text{m}^3 \text{h}^{-1}$)		0.56
Viral load, L ()		10^7
Viral emission rate, G (RNA copies per hour)		394
Community infection rate, C		1:100
Dose, D (viable virions inhaled)	0.245	2.450
REI	1	10

All values converted to SI units before application.

Table 3: Scenario inputs and calculations of population risk.

	Big Office Reference	Small Office Comparator
Viral load [36]		
()	N(7,1.4)	
$P(R)$ (%)	0.062	0.620
$P(I = 0)$ (%)	61	95
$P(0 < I < N)$ (%)	39	5
\bar{I}	1.27	1.02
$P(S)$ (%)	39	5
PPI (%)	1.59	0.43
TR		0.27

N, normal(μ, σ)
All values converted to SI units before application.

240 *2.1. Dose curve constant k*

241 Figure 1 shows that when the dose k values are low (< 50) then the PPI
242 begins to increase rapidly as lower doses are required to result in a significant
243 proportion of susceptibles in a scenario population becoming infected. The
244 rate of increase in PPI is greater in Big Office due to the larger population of
245 susceptibles. This results demonstrate that the dynamics of the dose response
246 are important in understanding the PPI and more work is required to better
247 understand these characteristics for SARS-CoV-2. Epidemiological evidence
248 can provide some illumination as to what bounds values of k with respect to
249 measured far-field transmission rates in indoor scenarios.

250 *2.2. Virion viability*

251 In our study, we take the conservative assumption that all viral genome
252 copies (RNA copies) in an inhaled dose are viable virions. The actual pro-
253 portion it more likely to be orders of magnitude lower, with estimates in the

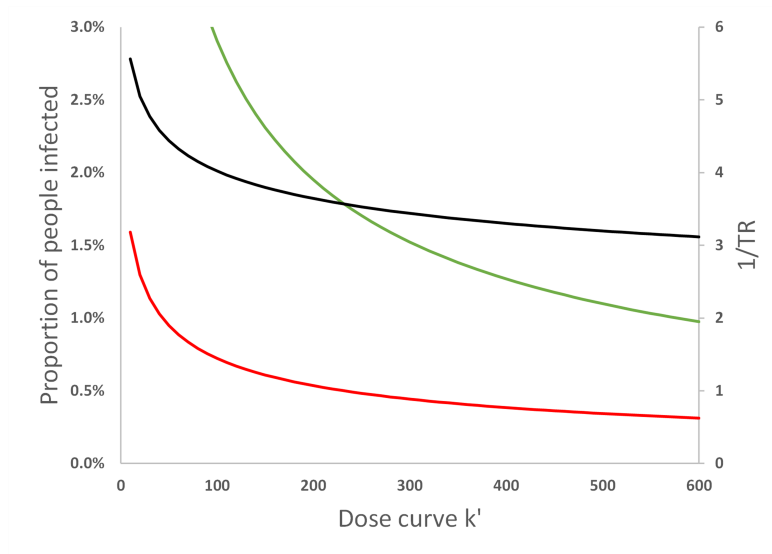


Figure 1: The effect of increasing the dose curve constant, k , on the Big Office PPI (green), Small Office PPI (red) and the TR (black). As k increases the size of the inhaled dose needed to give an equivalent probability of infection increases. All values are illustrative.

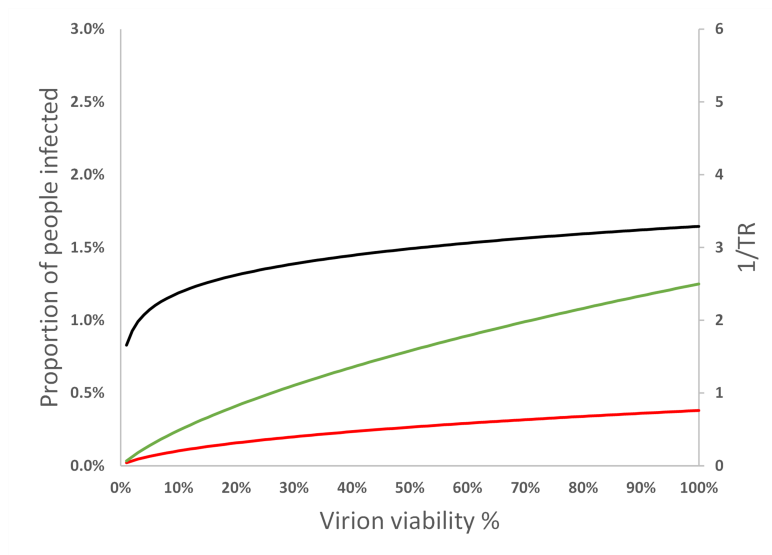


Figure 2: The effect of increasing the virion viability on the Big Office PPI (green), Small Office PPI (red) and the TR (black). As the proportion of RNA copies in the inhaled dose that are viable of virions increase, the probability of infection increases. All values are illustrative.

254 literature of a range of 1 : 100 to 1 : 10000 of viable virions to RNA copies.
 255 Miller *et al.* suggests that around 1 : 1000 genome copies are likely to be in-
 256 fectious virion whilst Adenaiye *et al.* suggest that from mid-turbinate swabs
 257 there is around 1 : 10⁴ viable virus per measured genome copies[14, 24].

258 Figure 2 shows the *PPI* in both Big Office and Small Office reduces as
 259 the proportion of viable virions decreases. Whilst the *TR* decreases, the
 260 ratio of Small Office to Big Office remains above 2, but the absolute values
 261 of *PPI* become very low as virion viability < 1%, suggesting that far-field
 262 transmission, given the assumptions in Tables 2 and 3, is very unlikely if
 263 virion viability is low.

264 *2.3. Viral Load of infected*

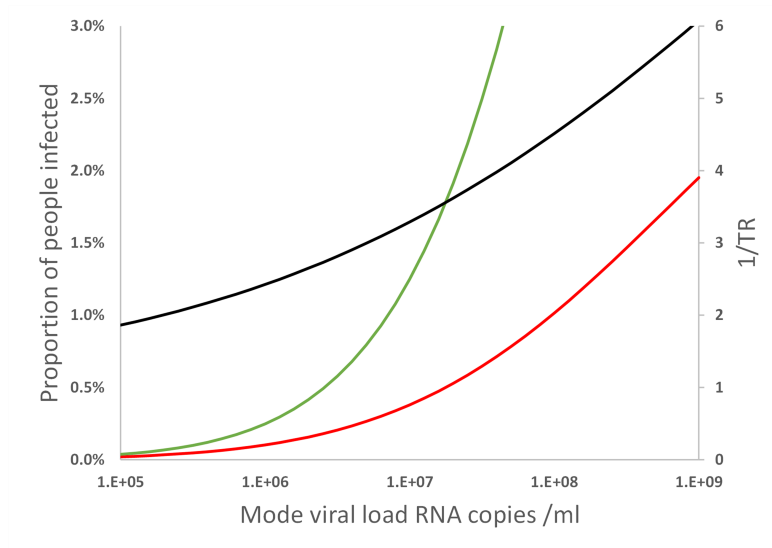


Figure 3: The effect of increasing the mode viral load of the infected population on the Big Office *PPI* (green), Small Office *PPI* (red) and the *TR* (black). As the mode viral load of the infectors increases, the emission rate of RNA copies increases, resulting in an increase in dose and *PPI*. All values are illustrative.

265 The inhaled dose is also a function of the viral load distribution within

266 the infected population. We assume that the log 10 viral load in RNA copies
 267 per ml is normally distributed with a mean of $\mu = 7$ and a standard deviation
 268 of $\sigma = 1.4$. Figure 3 shows the change in the probability of transmission and
 269 the TR when the mean log value of the distribution is varied between 5 and
 270 9. When the mean is low, the probability of one or more infectors having
 271 a sufficiently high emission rate to lead to the inhalation of an infective dose
 272 is very low, ie when $\mu = 5$. Conversely, increasing the probability of the
 273 infectors having a high viral load (by increasing μ) rapidly increases the
 274 probability of transmission in both scenarios, and an increase in the TR .

275 *2.4. Space Volume per person*

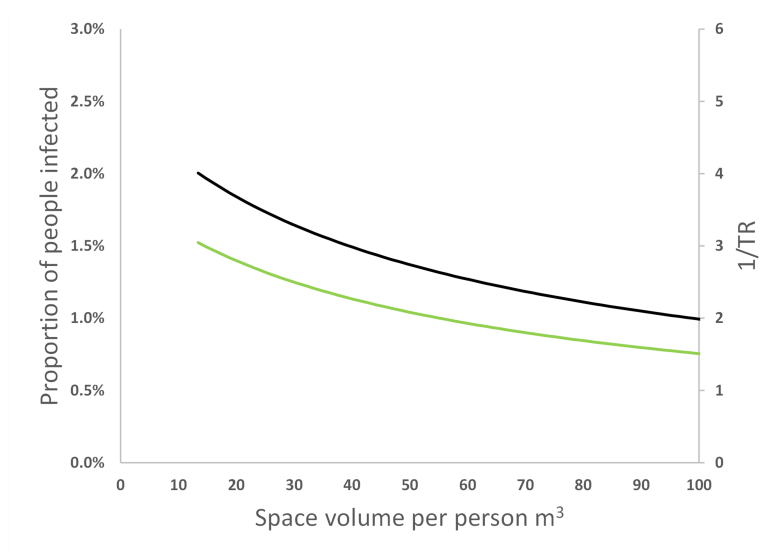


Figure 4: The effect of increasing the *per capita* space volume, V , in the Big Office on the PPI (green) and the TR (black) when the *per capita* space volume in the Small Office is constant. All values are illustrative.

276 Figure 4 shows that increasing the *per capita* space volume in the Big
 277 Office when the *per capita* space volume in the Small Office, while maintain-

278 ing a constant *per capita* ventilation in both spaces has a similar effect to
279 increasing the *per capita* ventilation. This is because the dose is inversely
280 proportional to volume. Furthermore, the product of the space volume and
281 the total removal rate, ϕV , is proportional to the concentration of the virus
282 in the air and, therefore, the dose. The *per capita* ventilation rate is constant
283 in both spaces and so the air change rate in the Big Office decreases as its
284 volume increases. However, this reduction is offset by the surface deposition
285 and biological decay rates, which remain constant and have a greater effect
286 on the value of the equivalent ventilation rate, ψV , as the space volume
287 increases.

288 Equation 1 assumes a steady-state concentration of the virus has been
289 reached based on the assumption that the exposure time, T , is significant.
290 However, the time taken to reach the steady-state concentration in large
291 spaces may be significant and affects the dose over shorter exposure periods.
292 This is an example of the *reservoir effect*, the ability of indoor air to act as
293 a fresh-air reservoir and absorb the impact of contaminant emissions. The
294 greater the space volume, the greater the effect. These factors highlight the
295 benefits of increasing the *per capita* space volume.

296 2.5. Exposure Time

297 Increasing exposure time when an infected person is present in the space
298 for a significant period of time the exponent of Equation 1 becomes relatively
299 small so that $e^{-\phi T} \rightarrow 0$ and the inhaled dose is approximately proportional
300 to the exposure time, however, the effect of the dose curve relationship means
301 that *PPI* is not directly proportional to exposure time. Reducing the expo-
302 sure time from 12 to 4 hours will reduce the probability of an inhaled dose

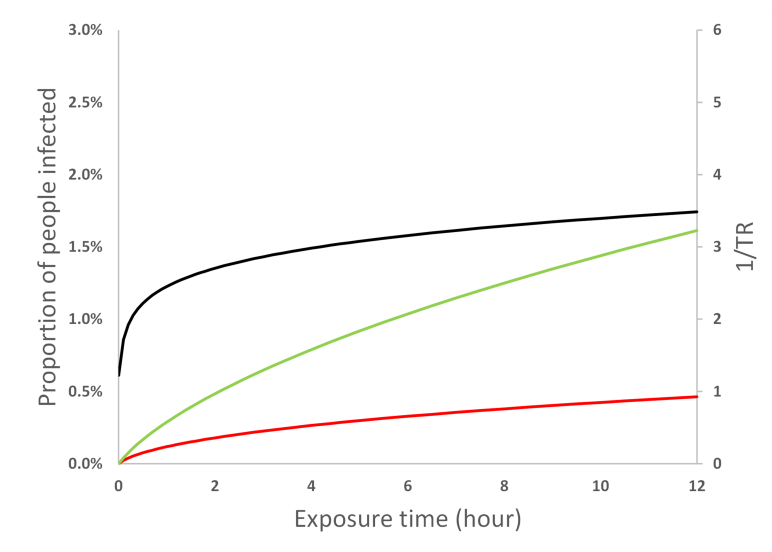


Figure 5: The effect of increasing the exposure time on the Big Office PPI (green), Small Office PPI (red) and the TR (black). As the exposure time increases, the dose increases, resulting in an increase in PPI . All values are illustrative.

303 leading to infection from relatively low viral load infectors, but will have less
 304 effect on the higher viral load infectors. It is only when exposure times be-
 305 come very short that the PPI reduces rapidly due to the reduced probability
 306 of even the higher viral load infectors delivering a dose likley to lead to infec-
 307 tion, (as this study assumes steady state, this rate of reduction will be more
 308 pronounced when considering the reservoir effect) this is consistent with the
 309 findings of Miller *et al.* in the requirement for reduced exposure time, as well
 310 as improved ventilation to significantly reduce the risk of transmission in the
 311 case of the Skagit choir superspreading event [14].

312 **3. Rapid Antigen Testing**

313 Lateral flow testing uses a rapid lateral flow device (LFD) based on col-
 314 loidal gold immunochromatography designed to detect the presence of SARS-

315 CoV-2 nucleocapsid antigens in nasopharyngeal swabs. These tests are not
316 as sensitive as PCR tests in detecting the presence of SARS-CoV-2, but they
317 have been demonstrated to have a good ability at detecting higher viral loads
318 in infectors [37, 38]. Because the results of our analysis demonstrates that
319 the higher viral emissions are responsible for the greater *PPI* we consider
320 the effect of a scenario where widespread adoption of LFD use is success-
321 ful in identifying individuals with high viral load and removing them from
322 the scenario. The distribution of log 10 viral loads of infectors is assumed
323 to be normally distributed with a mean of $\mu = 7$ and a standard devia-
324 tion of $\sigma = 1.4$, then the proportion of individuals with viral loads greater
325 than 10^9 RNA copies per ml is about 9%. We can assume a proportion of
326 these are removed from the Small Office and Big Office scenarios and con-
327 sider the effects on the *PPI* given the assumptions in Table 2. The prob-
328 ability of viral load, $P(L)$ when $VL > 10^9$ RNA copies/ml is multiplied by
329 $1 - LFD_{effectiveness}$ where the effectiveness is assumed to be 70%, Figure 6.
330 These results show that LFD could be an effective measure to reduce the
331 *PPI*, reducing both the absolute *PPI* in Big Office (from 1.59 to 0.60) and
332 Small Office (from 0.43 to 0.22), as well as the *TR*. However, with a *C* of
333 1 : 100, persons with a viral load greater than 10^9 RNA copies/ml represent
334 around 0.09% of the total population, so although LFD could be an effective
335 method of removing the highest viral loads from a scenario, a lot of lateral
336 flow tests need to be conducted to capture every high viral load infector.

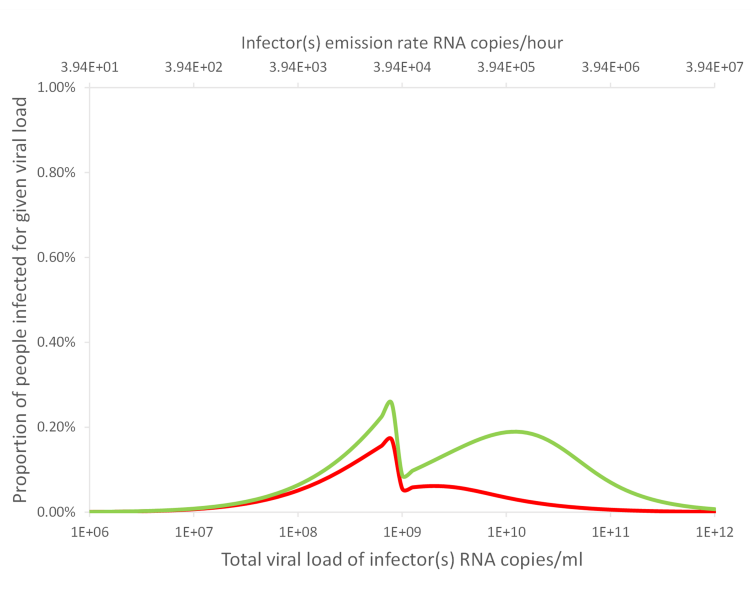


Figure 6: An indication of the relationship between the proportion of a population infected for a particular viral load when the community infection rate is $C = 1\%$ and where LFD that are 70% effective at removing infectors with viral loads greater than 10^9 RNA copies per ml. The area under the curve represents the total proportion of people infected for the Small Office (red) and the Big Office (green). All values are illustrative.

337 **4. Alternative assumptions, lower virion viability and higher mode**
338 **viral load**

339 As detailed previously, the assumptions used in the main paper with
340 respect to how RNA copies represent viable virions is highly conservative,
341 and so below we use a more realistic value of 1% RNA copies as representing
342 viable virions. Additionally we also consider that the mode value for viral
343 load is 10^8 RNA copies per ml, to represent a potential increase in viral load
344 that could be the result of a variant of SARS-CoV-2, see Tables 4 and 5.

345 Figure 7 shows how the reduction in virion viability shifts the Big Office
346 and Small Office dose curves to the right of the graph as greater viral emission
347 is required to result in a dose of viable virion likely to give rise to an infection.
348 The viral load and the probability that a single has that viral load, $P(L)$,
349 is also shifted to the right. The dashed vertical lines show the viral load
350 required to give a 50% probability that the dose will lead to an infection for
351 each scenario, $P(R) = 50\%$. The area under the blue curve to the right of
352 each vertical line is the probability that the viral load of the infected person
353 leads to $P(R) \geq 50\%$. The probability is much smaller for the Big Office,
354 which has the lower REI. This probability that an infected person has a viral
355 load that leads to $P(R) \geq 50\%$ is small, suggesting that the most likely
356 outcome is $P(R) \leq 50\%$.

357 The effect of varying assumptions on the shape of the plotted PPI curves
358 have been described in detail in the main paper and above 2. In Figures 8
359 and 9 the affect on PPI and TR is shown for the assumptions made in the
360 main paper (on the left) with the higher modal viral load and lower virion
361 viability (on the right). The effect on the absolute values is pronounced due

362 to the reduced virion viability assumption, and given these assumptions the
363 PPI is very low, suggesting that far field transmission is likely to be rare and
364 efforts taken to minimise TR should consider the absolute improvements in
365 PPI when assessing the benefits in TR reduction compared to the costs of,
366 for example, the increased energy use needed to increase ventilation above
367 current guidance. It is important to note how changes in assumptions needed
368 to estimate viral emission rates have large impacts on absolute values of PPI
369 and thus the uncertainty in these assumptions needs to be considered when
370 interpreting comparisons.

Table 4: Scenario inputs and calculations of individual risk.

	Big Office Reference	Small Office Comparator
Number of occupants, N	50	5
Space Volume, V (m^3)	1500	150
<i>Per capita</i> volume, $V N^{-1}$ (m^3 per person)	30	30
Air flow rate, ψV ($l s^{-1}$)	500	50
Air change rate, ψ (h^{-1})	1.2	1.2
Removal rate, ϕ (h^{-1})	2.26	2.26
Equivalent ventilation rate, ϕV ($l s^{-1}$)	942	94.2
Exposure time, T (h)	8	8
Dose constant, k [32]	410	410
Viable fraction, v (%)	1	1
Viral load (RNA copies per ml) [36]	10^8	10^8
Respiratory activity, <i>breathing: talking</i> (%)	75:25	75:25
Viral emission rate, G (RNA copies per hour)	394	394
Respiratory rate, q_{sus} ($m^3 h^{-1}$)	0.56	0.56
Community infection rate, C	1:100	1:100
Dose, D (viable virions inhaled)	0.002	0.025
REI	1	10

All values converted to SI units before application.

371 For this analysis we make the assumption that 1% of genome copies

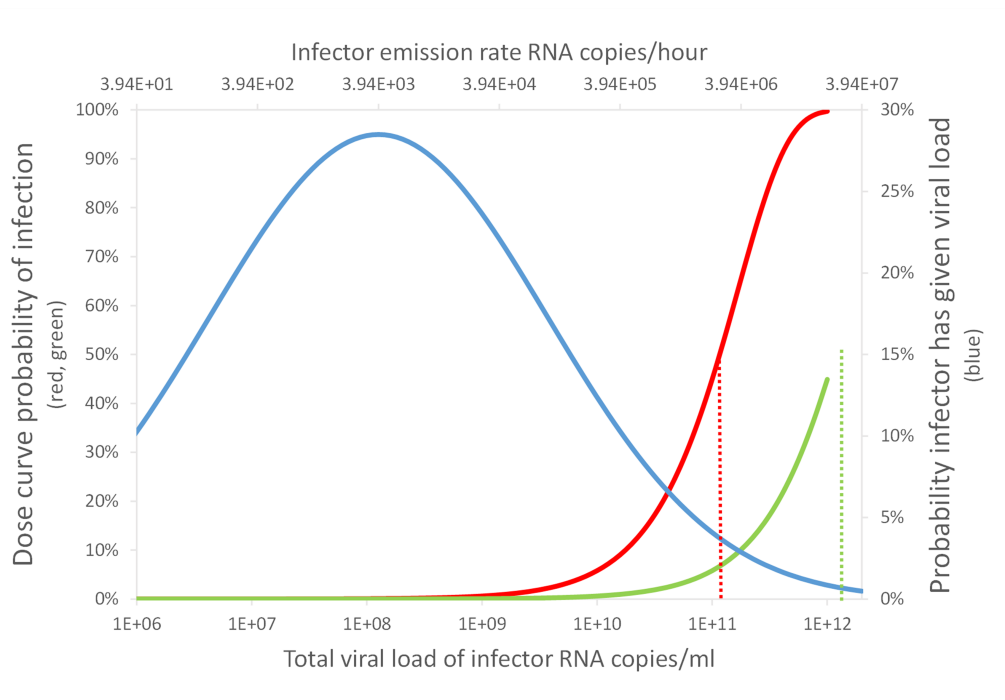


Figure 7: An indication of the relationship between the viral load, L , and the consequent probability of infection, $P(R)$, in the Big Office (green) and Small Office (red) for a susceptible occupant, and the probability of a single infected person having a viral load, $P(L)$, (blue). Dashed vertical lines indicate the viral load required for $P(R) = 50\%$.

Table 5: Scenario inputs and calculations of population risk.

	Big Office Reference	Small Office Comparator
Viral load [36] (RNA copies per ml)	LN ($2.7 \times 10^{10}, 3.6 \times 10^{11}$)	
$P(R)$ (%)	0.062	0.620
$P(I = 0)$ (%)	61	95
$P(0 < I < N)$ (%)	39	5
\bar{I}	1.27	1.02
$P(S)$ (%)	39	5
PPI (%)	0.241	0.119
TR		0.49

LN, log-normal(μ, σ)
 All values converted to SI units before application.

372 (RNA copies) represent viable virions, which is a more realistic magnitude to
 373 assume given the literature

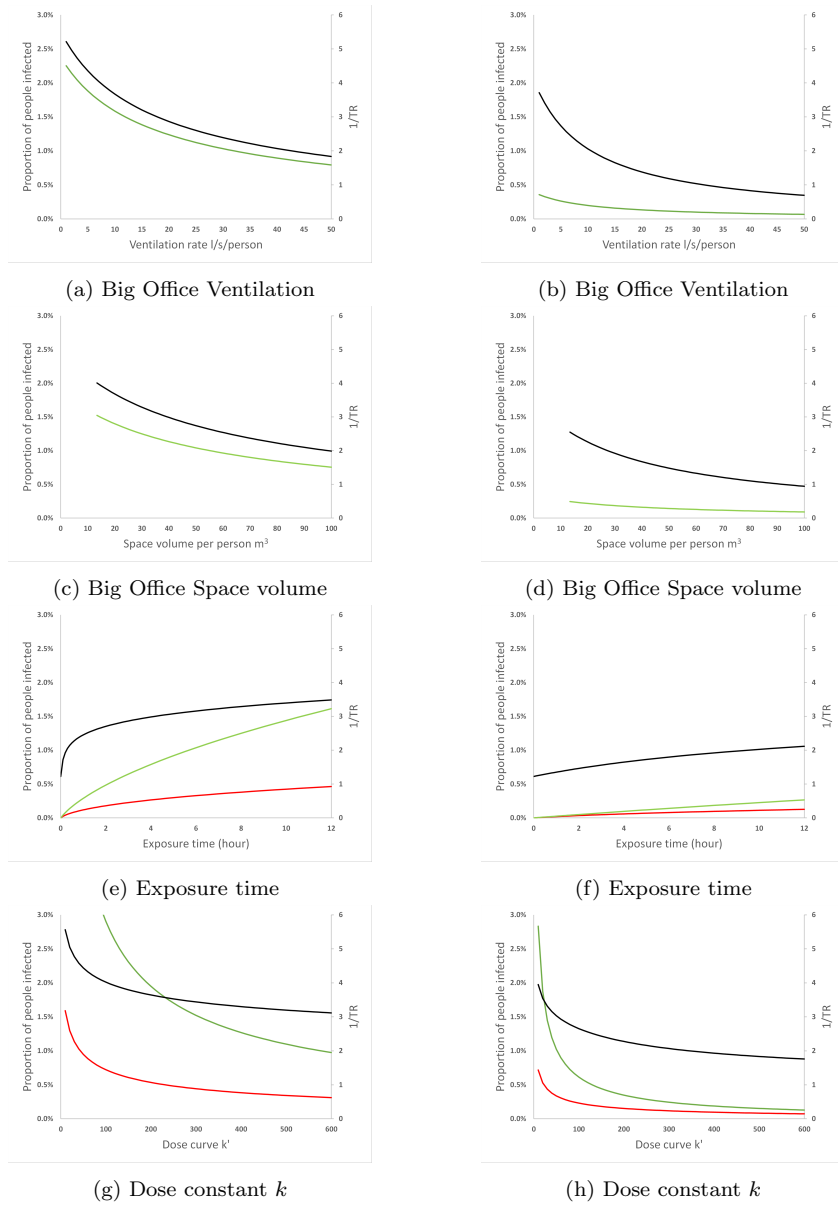


Figure 8: The effect of modulating assumption values on the Big Office PPI (green), Small Office PPI (red) and the TR (black). Left hand images the modal viral load assumed to be 10^7 RNA copies per ml and virion viability 100%, right hand images the modal viral load assumed to be 10^8 RNA copies per ml and virion viability 1%. All values are illustrative.

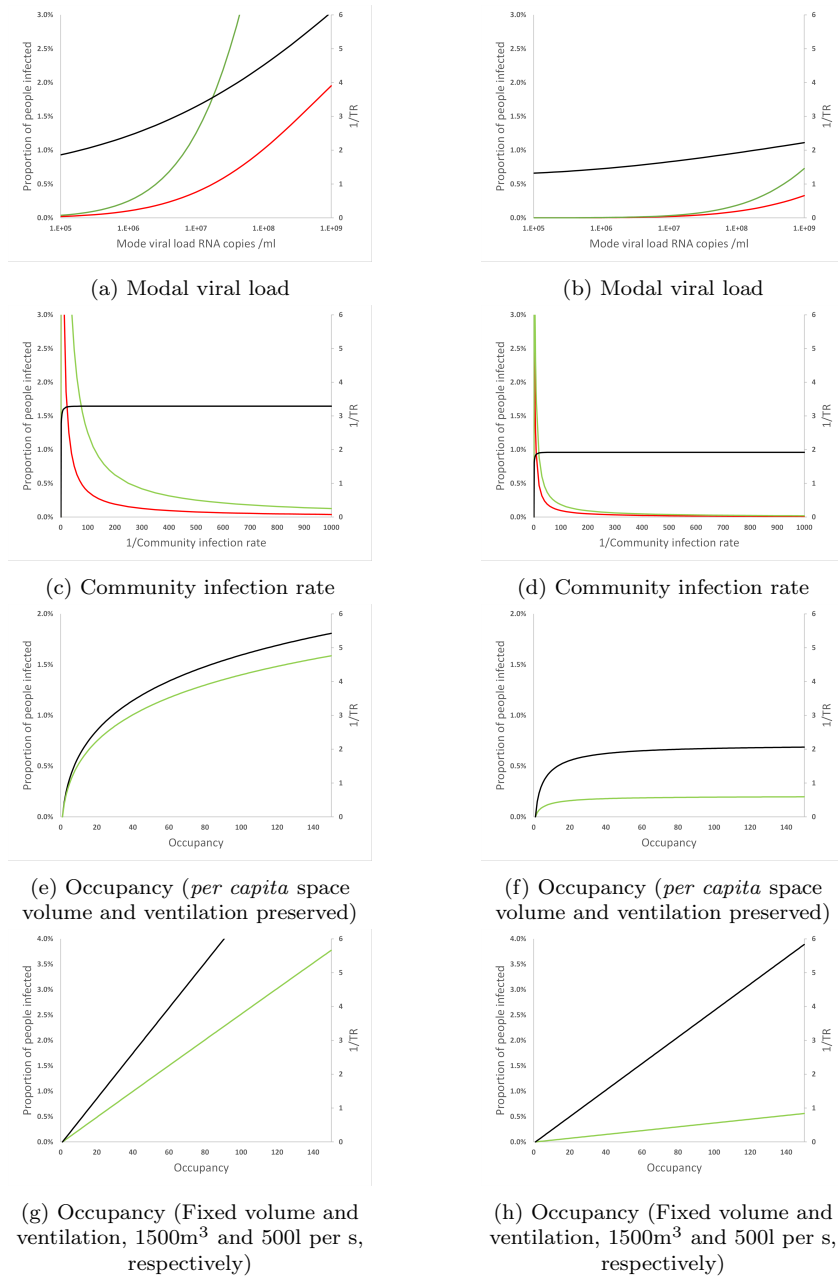


Figure 9: The effect of modulating assumption values on the Big Office *PPI* (green), Small Office *PPI* (red) and the *TR* (black). Left hand images the modal viral load assumed to be 10^7 RNA copies per ml and virion viability 100%, right hand images the modal viral load assumed to be 10^8 RNA copies per ml and virion viability 1%. All values are illustrative.

374 **5. Equations**

375 See main text for explanations of the equations.

376 *5.1. Inhaled Dose*

$$D \simeq \frac{K q_{sus} G T v}{\phi V} \quad (1)$$

377 *5.2. Dose response curve*

$$P(R) = 1 - e^{-D/k} \quad (2)$$

378 **Acknowledgements**

379 The authors acknowledge the Engineering and Physical Sciences Research
380 Council (EP/W002779/1) who financially supported this work. They are also
381 grateful to Constanza Molina for her comments on this paper.

382 **References**

- 383 [1] T. Watanabe, T. A. Bartrand, M. H. Weir, T. Omura, C. N. Haas,
384 Development of a dose-response model for sars coronavirus. risk analysis:
385 an official publication of the society for risk analysis, Risk Anal 30 (7)
386 (2010) 1129–1138. doi:10.1111/j.1539-6924.2010.01427.x.
- 387 [2] H. Parhizkar, K. G. Van Den Wymelenberg, C. N. Haas, R. L. Corsi, A
388 Quantitative Risk Estimation Platform for Indoor Aerosol Transmission of
389 COVID-19, Risk Analysis 0 (0) (2021). doi:10.1111/risa.13844.
- 390 [3] X. Zhang, J. Wang, Dose-response Relation Deduced for Coronaviruses
391 From Coronavirus Disease 2019, Severe Acute Respiratory Syndrome,
392 and Middle East Respiratory Syndrome: Meta-analysis Results and
393 its Application for Infection Risk Assessment of Aerosol Transmission,
394 Clinical Infectious Diseases (Xx Xxxx) (2020) 1–5. doi:10.1093/cid/
395 ciaa1675.
- 396 [4] J. Schijven, L. C. Vermeulen, A. Swart, A. Meijer, E. Duizer, A. M.
397 de Roda Husman, Quantitative microbial risk assessment for airborne
398 transmission of sars-cov-2 via breathing, speaking, singing, coughing,
399 and sneezing, Environmental Health Perspectives 129 (4) (2021) 1–10.
400 doi:10.1289/EHP7886.
- 401 [5] M. Cevik, K. Kuppalli, J. Kindrachuk, M. Peiris, Virology, transmission,
402 and pathogenesis of SARS-CoV-2, The BMJ 371 (2020) 1–6. doi:10.
403 1136/bmj.m3862.

- 404 [6] M. Cevik, M. Tate, O. Lloyd, A. E. Maraolo, J. Schafers, A. Ho,
405 SARS-CoV-2, SARS-CoV, and MERS-CoV viral load dynamics, du-
406 ration of viral shedding, and infectiousness: a systematic review and
407 meta-analysis, *The Lancet Microbe* 2 (1) (2021) e13–e22. doi:10.1016/
408 S2666-5247(20)30172-5.
409 URL [http://dx.doi.org/10.1016/S2666-5247\(20\)30172-5](http://dx.doi.org/10.1016/S2666-5247(20)30172-5)
- 410 [7] Y. Pan, D. Zhang, P. Yang, L. L. Poon, Q. Wang, Viral load of SARS-
411 CoV-2 in clinical samples, *The Lancet Infectious Diseases* 20 (4) (2020)
412 411–412. doi:10.1016/S1473-3099(20)30113-4.
413 URL [http://dx.doi.org/10.1016/S1473-3099\(20\)30113-4](http://dx.doi.org/10.1016/S1473-3099(20)30113-4)
- 414 [8] S. Karimzadeh, R. Bhopal, H. Nguyen Tien, Review of infective
415 dose, routes of transmission and outcome of COVID-19 caused
416 by the SARS-COV-2: comparison with other respiratory viruses–
417 CORRIGENDUM, *Epidemiology and Infection* 149 (2021) e116.
418 doi:10.1017/S0950268821001084.
419 URL [https://www.cambridge.org/core/product/identifier/
420 S0950268821001084/type/journal_article](https://www.cambridge.org/core/product/identifier/S0950268821001084/type/journal_article)
- 421 [9] R. Challen, E. Brooks-Pollock, J. M. Read, L. Dyson, K. Tsaneva-
422 Atanasova, L. Danon, Risk of mortality in patients infected with SARS-
423 CoV-2 variant of concern 202012/1: Matched cohort study, *The BMJ*
424 372 (2021) 1–10. doi:10.1136/bmj.n579.
- 425 [10] R. Ke, P. P. Martinez, R. L. Smith, L. L. Gibson, A. Mirza, M. Conte,
426 N. Gallagher, C. H. Luo, J. Jarrett, A. Conte, M. Farjo, K. K. O.
427 Walden, G. Rendon, C. J. Fields, R. Fredrickson, D. C. Edmonson,

- 428 M. E. Baughman, K. K. Chiu, J. Yedetore, J. Quicksall, A. N. Owens,
429 J. Broach, Daily sampling of early SARS-CoV-2 infection reveals
430 substantial heterogeneity in infectiousness, medRxiv (July 12th 2021)
431 (2021) 1–23. doi:<https://doi.org/10.1101/2021.07.12.21260208>.
432 URL [https://www.medrxiv.org/content/10.1101/2021.07.12.](https://www.medrxiv.org/content/10.1101/2021.07.12.21260208v1)
433 [21260208v1](https://www.medrxiv.org/content/10.1101/2021.07.12.21260208v1)
- 434 [11] A. S. Walker, E. Pritchard, T. House, J. V. Robotham, P. J. Birrell,
435 I. Bell, J. Bell, J. Newton, J. Farrar, I. Diamond, R. Studley, J. Hay,
436 K.-D. Vihta, T. E. Peto, N. Stoesser, P. C. Matthews, D. W. Eyre,
437 K. Pouwels, Ct threshold values, a proxy for viral load in community
438 SARS-CoV-2 cases, demonstrate wide variation across populations and
439 over time, eLife 10 (jul 2021). doi:[10.7554/eLife.64683](https://doi.org/10.7554/eLife.64683).
440 URL <https://elifesciences.org/articles/64683>
- 441 [12] P. Z. Chen, N. Bobrovitz, Z. Premji, M. Koopmans, D. N. Fisman, F. X.
442 Gu, Heterogeneity in transmissibility and shedding SARS-CoV-2 via
443 droplets and aerosols, eLife 10 (2021) 1–32. doi:[10.7554/eliflife.65774](https://doi.org/10.7554/eliflife.65774).
- 444 [13] G. Buonanno, L. Stabile, L. Morawska, Estimation of airborne viral
445 emission: Quanta emission rate of SARS-CoV-2 for infection risk as-
446 sessment, Environment International 141 (May) (2020) 105794. doi:
447 [10.1016/j.envint.2020.105794](https://doi.org/10.1016/j.envint.2020.105794).
- 448 [14] S. L. Miller, W. W. Nazaroff, J. L. Jimenez, A. Boerstra, S. J. Dancer,
449 J. Kurnitski, L. C. Marr, L. Morawska, C. Noakes, Transmission of
450 SARS-CoV-2 by inhalation of respiratory aerosol in the Skagit Valley

- 451 Chorale superspreading event, *Indoor Air* in press (2020). doi:doi.
452 org/10.1111/ina.12751.
- 453 [15] J. Yan, M. Grantham, J. Pantelic, P. J. B. De Mesquita, B. Albert,
454 F. Liu, S. Ehrman, D. K. Milton, Infectious virus in exhaled breath of
455 symptomatic seasonal influenza cases from a college community, *Pro-*
456 *ceedings of the National Academy of Sciences of the United States of*
457 *America* 115 (5) (2018) 1081–1086. doi:10.1073/pnas.1716561115.
- 458 [16] Q. Yang, T. K. Saldi, P. K. Gonzales, E. Lasda, C. J. Decker, K. L.
459 Tat, M. R. Fink, C. R. Hager, J. C. Davis, C. D. Ozeroff, D. Muhrad,
460 S. K. Clark, W. T. Fattor, N. R. Meyerson, C. L. Paige, A. R. Gilchrist,
461 A. Barbachano-Guerrero, E. R. Worden-Sapper, S. S. Wu, G. R. Bris-
462 son, M. B. McQueen, R. D. Dowell, L. Leinwand, R. Parker, S. L.
463 Sawyer, Just 2 percent of sars-cov-2 positive individuals carry 90 per-
464 cent of the virus circulating in communities, *Proceedings of the National*
465 *Academy of Sciences* 118 (21) (2021) e2104547118. doi:10.1073/pnas.
466 2104547118.
467 URL <http://www.pnas.org/lookup/doi/10.1073/pnas.2104547118>
- 468 [17] D. K. Milton, M. P. Fabian, B. J. Cowling, M. L. Grantham, J. J.
469 McDevitt, Influenza Virus Aerosols in Human Exhaled Breath: Particle
470 Size, Culturability, and Effect of Surgical Masks, *PLoS Pathogens* 9 (3)
471 (2013). doi:10.1371/journal.ppat.1003205.
- 472 [18] P. Fabian, J. J. McDevitt, W. H. DeHaan, R. O. Fung, B. J. Cowling,
473 K. H. Chan, G. M. Leung, D. K. Milton, Influenza virus in human

- 474 exhaled breath: An observational study, PLoS ONE 3 (7) (2008) 5–10.
475 doi:10.1371/journal.pone.0002691.
- 476 [19] K. K. Coleman, D. J. W. Tay, K. S. Tan, S. W. X. Ong, T. S. Than,
477 M. H. Koh, Y. Q. Chin, H. Nasir, T. M. Mak, J. J. H. Chu, D. K. Milton,
478 V. T. K. Chow, P. A. Tambyah, M. Chen, K. W. Tham, Viral Load of
479 Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) in
480 Respiratory Aerosols Emitted by Patients With Coronavirus Disease
481 2019 (COVID-19) While Breathing, Talking, and Singing, Clinical
482 Infectious Diseases 2 (Xx) (2021) 1–7. doi:10.1093/cid/ciab691.
483 URL [https://academic.oup.com/cid/advance-article/doi/10.](https://academic.oup.com/cid/advance-article/doi/10.1093/cid/ciab691/6343417)
484 [1093/cid/ciab691/6343417](https://academic.oup.com/cid/advance-article/doi/10.1093/cid/ciab691/6343417)
- 485 [20] F. K. Gregson, N. A. Watson, C. M. Orton, A. E. Haddrell, L. P. Mc-
486 Carthy, T. J. Finnie, N. Gent, G. C. Donaldson, P. L. Shah, J. D.
487 Calder, B. R. Bzdek, D. Costello, J. P. Reid, Comparing aerosol concen-
488 trations and particle size distributions generated by singing, speaking
489 and breathing, Aerosol Science and Technology 55 (6) (2021) 681–691.
490 doi:10.1080/02786826.2021.1883544.
491 URL <https://doi.org/10.1080/02786826.2021.1883544>
- 492 [21] G. R. Johnson, L. Morawska, The mechanism of breath aerosol forma-
493 tion, Journal of Aerosol Medicine and Pulmonary Drug Delivery 22 (3)
494 (2009) 229–237. doi:10.1089/jamp.2008.0720.
- 495 [22] G. R. Johnson, L. Morawska, Z. D. Ristovski, M. Hargreaves,
496 K. Mengersen, C. Y. Chao, M. P. Wan, Y. Li, X. Xie, D. Kato-
497 shevski, S. Corbett, Modality of human expired aerosol size distri-

- 498 butions, *Journal of Aerosol Science* 42 (12) (2011) 839–851. doi:
499 10.1016/j.jaerosci.2011.07.009.
- 500 [23] L. Morawska, G. R. Johnson, Z. D. Ristovski, M. Hargreaves,
501 K. Mengersen, S. Corbett, C. Y. Chao, Y. Li, D. Katoshevski, Size
502 distribution and sites of origin of droplets expelled from the human res-
503 piratory tract during expiratory activities, *Journal of Aerosol Science*
504 40 (3) (2009) 256–269. doi:10.1016/j.jaerosci.2008.11.002.
- 505 [24] O. O. Adenaiye, J. Lai, P. J. B. de Mesquita, F. Hong, S. Youssefi,
506 J. German, S.-H. S. Tai, B. Albert, M. Schanz, S. Weston, J. Hang,
507 C. Fung, H. K. Chung, K. K. Coleman, N. Sapoval, T. Treangen,
508 I. M. Berry, K. Mullins, M. Frieman, T. Ma, D. K. Milton, Infec-
509 tious SARS-CoV-2 in Exhaled Aerosols and Efficacy of Masks Dur-
510 ing Early Mild Infection, *medRxiv* (2021) 1–19arXiv:2021.08.13.
511 21261989, doi:<https://doi.org/10.1101/2021.08.13.21261989>.
512 URL <https://doi.org/10.1101/2021.08.13.21261989>
- 513 [25] G. Buonanno, L. Morawska, L. Stabile, Quantitative assessment of the
514 risk of airborne transmission of SARS-CoV-2 infection: Prospective and
515 retrospective applications, *Environment International* 145 (June) (2020)
516 106112. doi:10.1016/j.envint.2020.106112.
517 URL <https://doi.org/10.1016/j.envint.2020.106112>
- 518 [26] A. C. Fears, W. B. Klimstra, P. Duprex, A. Hartman, S. C. Weaver,
519 K. S. Plante, D. Mirchandani, J. A. Plante, P. V. Aguilar, D. Fernández,
520 A. Nalca, A. Totura, D. Dyer, B. Kearney, M. Lackemeyer, J. K. Bo-
521 hannon, R. Johnson, R. F. Garry, D. S. Reed, C. J. Roy, Persistence

- 522 of Severe Acute Respiratory Syndrome Coronavirus 2 in Aerosol Sus-
523 pensions, *Emerging infectious diseases* 26 (9) (2020). doi:10.3201/
524 eid2609.201806.
- 525 [27] B. Jones, P. Sharpe, C. Iddon, E. A. Hathway, C. J. Noakes, S. Fitzger-
526 ald, Modelling uncertainty in the relative risk of exposure to the SARS-
527 CoV-2 virus by airborne aerosol transmission in well mixed indoor air,
528 *Building and Environment* 191 (October 2020) (2021) 107617. doi:
529 10.1016/j.buildenv.2021.107617.
530 URL <https://doi.org/10.1016/j.buildenv.2021.107617>
- 531 [28] J. Lelieveld, F. Helleis, S. Borrmann, Y. Cheng, F. Drewnick, G. Haug,
532 T. Klimach, J. Sciare, H. Su, U. Pöschl, Model calculations of aerosol
533 transmission and infection risk of covid-19 in indoor environments, *Inter-
534 national Journal of Environmental Research and Public Health* 17 (21)
535 (2020) 1–18. doi:10.3390/ijerph17218114.
- 536 [29] P. Y. Chia, K. K. Coleman, Y. K. Tan, S. Wei, X. Ong, M. Gum, S. K.
537 Lau, X. F. Lim, A. S. Lim, S. Sutjipto, P. H. Lee, T. T. Son, B. E. Young,
538 D. K. Milton, G. C. Gray, S. Schuster, T. Barkham, P. P. De, S. Vasoo,
539 M. Chan, B. Sze, P. Ang, Detection of air and surface contamination by
540 SARS-CoV-2 in hospital rooms of infected patients, *Nature Communi-
541 cations* 11 (2800) (2020). doi:10.1038/s41467-020-16670-2.
- 542 [30] J. Ma, X. Qi, H. Chen, X. Li, Z. Zhang, H. Wang, L. Sun, L. Zhang,
543 J. Guo, L. Morawska, S. A. Grinshpun, P. Biswas, R. C. Flagan, M. Yao,
544 Coronavirus Disease 2019 Patients in Earlier Stages Exhaled Millions of

- 545 Severe Acute Respiratory Syndrome Coronavirus 2 Per Hour, *Clinical in-*
546 *fectious diseases : an official publication of the Infectious Diseases Soci-*
547 *ety of America* 72 (10) (2021) e652–e654. doi:10.1093/cid/ciaa1283.
- 548 [31] V. M. Corman, I. Eckerle, T. Bleicker, A. Zaki, O. Landt, M. Eschbach-
549 Bludau, S. van Boheemen, R. Gopal, M. Ballhause, T. M. Bestebroer,
550 D. Muth, M. A. Müller, J. F. Drexler, M. Zambon, A. D. Osterhaus,
551 R. M. Fouchier, C. Drosten, Detection of a novel human coronavirus by
552 real-time reverse-transcription polymerase chain reaction, *Eurosurveil-*
553 *lance* 17 (39) (2012) 1–6. doi:10.2807/ese.17.39.20285-en.
554 URL <http://dx.doi.org/10.2807/ese.17.39.20285-en>
- 555 [32] M. L. DeDiego, L. Pewe, E. Alvarez, M. T. Rejas, S. Perlman, L. En-
556 juanes, Pathogenicity of severe acute respiratory coronavirus deletion
557 mutants in hACE2 transgenic mice, *Virology* 376 (2) (2008) 379–389.
558 doi:<https://doi.org/10.1016/j.virol.2008.03.005>.
559 URL [https://www.sciencedirect.com/science/article/pii/](https://www.sciencedirect.com/science/article/pii/S004268220800175X)
560 [S004268220800175X](https://www.sciencedirect.com/science/article/pii/S004268220800175X)
- 561 [33] F. Aiano, A. A. Mensah, K. McOwat, C. Obi, A. Vusirikala, A. A.
562 Powell, J. Flood, J. Bosowski, L. Letley, S. Jones, Z. Amin-Chowdhury,
563 J. Lacy, I. Hayden, S. A. Ismail, M. E. Ramsay, S. N. Ladhani, V. Saliba,
564 COVID-19 outbreaks following full reopening of primary and secondary
565 schools in England: Cross-sectional national surveillance, November
566 2020, *The Lancet Regional Health - Europe* 6 (May 2020) (2021) 100120.
567 doi:10.1016/j.lanepe.2021.100120.
- 568 [34] ONS, ONS Coronavirus (COVID-19) Infection Survey.

- 569 URL [https://www.ons.gov.uk/peoplepopulationandcommunity/](https://www.ons.gov.uk/peoplepopulationandcommunity/healthandsocialcare/conditionsanddiseases/bulletins/coronaviruscovid19infectionsurveypilot/previousReleases)
570 [healthandsocialcare/conditionsanddiseases/bulletins/](https://www.ons.gov.uk/peoplepopulationandcommunity/healthandsocialcare/conditionsanddiseases/bulletins/coronaviruscovid19infectionsurveypilot/previousReleases)
571 [coronaviruscovid19infectionsurveypilot/previousReleases](https://www.ons.gov.uk/peoplepopulationandcommunity/healthandsocialcare/conditionsanddiseases/bulletins/coronaviruscovid19infectionsurveypilot/previousReleases)
- 572 [35] G. N. Sze To, C. Y. H. Chao, Review and comparison between the
573 wells–riley and dose-response approaches to risk assessment of infectious
574 respiratory diseases, *Indoor air* 20 (1) (2010) 2–16. doi:10.1111/j.
575 1600-0668.2009.00621.x.
- 576 [36] P. Z. Chen, N. Bobrovitz, Z. Premji, M. Koopmans, D. N. Fisman, F. X.
577 Gu, Sars-cov-2 shedding dynamics across the respiratory tract, sex, and
578 disease severity for adult and pediatric covid-19, *eLife* 10 (2021) e70458.
579 doi:10.7554/eLife.70458.
580 URL <https://doi.org/10.7554/eLife.70458>
- 581 [37] J. Ferguson, S. Dunn, A. Best, J. Mirza, B. Percival, M. Mayhew,
582 O. Megram, F. Ashford, T. White, E. Moles-Garcia, L. Crawford,
583 T. Plant, A. Bosworth, M. Kidd, A. Richter, J. Deeks, A. McNally,
584 Validation testing to determine the sensitivity of lateral flow testing for
585 asymptomatic SARSCoV-2 detection in low prevalence settings: Test-
586 ing frequency and public health messaging is key, *PLoS Biology* 19 (4)
587 (2021) 1–9. doi:10.1371/journal.pbio.3001216.
588 URL <http://dx.doi.org/10.1371/journal.pbio.3001216>
- 589 [38] L. Y. W. Lee, S. Rozmanowski, M. Pang, A. Charlett, C. Anderson,
590 G. J. Hughes, M. Barnard, L. Peto, R. Vipond, A. Sienkiewicz,
591 S. Hopkins, J. Bell, D. W. Crook, N. Gent, A. S. Walker, T. E. A.
592 Peto, D. W. Eyre, Severe Acute Respiratory Syndrome Coronavirus

593 2 (SARS-CoV-2) Infectivity by Viral Load, S Gene Variants and
594 Demographic Factors, and the Utility of Lateral Flow Devices to
595 Prevent Transmission, *Clinical Infectious Diseases* 6 (2021) 1–32.
596 doi:10.1093/cid/ciab421.
597 URL [https://academic.oup.com/cid/advance-article/doi/10.](https://academic.oup.com/cid/advance-article/doi/10.1093/cid/ciab421/6273394)
598 [1093/cid/ciab421/6273394](https://academic.oup.com/cid/advance-article/doi/10.1093/cid/ciab421/6273394)