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1 Increased airborne transmission of COVID-19 with new variants,
2 Implications for health policies

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32 **Abstract**

33 New COVID-19 variants, either of higher viral load such as delta or higher contagiousness like
34 omicron, can lead to higher airborne transmission than historical strains. This paper highlights their
35 implications for health policies, based on a clear analytical understanding and modeling of the
36 airborne contamination paths, of the dose following exposure, and the importance of the counting
37 unit for pathogens, itself linked to the dose-response law. Using the counting unit of Wells, i.e. the
38 quantum of contagium, we develop the conservation equation of quanta which allows deriving the
39 value of the quantum concentration at steady state for a well-mixed room. The link with the
40 monitoring concentration of carbon dioxide is made and used for a risk analysis of a variety of
41 situations for which we collected CO₂ time-series observations. The main conclusions of these
42 observations are that 1) the present norms of ventilation, are both insufficient and not respected,
43 especially in a variety of public premises, leading to high risk of contamination and that 2) air can
44 often be considered well-mixed. Finally, we insist that public health policy in the field of airborne
45 transmission should be based on a multi parameter analysis such as the time of exposure, the
46 quantum production rate, mask wearing and the infector proportion in the population in order to
47 evaluate the risk, considering the whole complexity of dose evaluation. Recognizing airborne
48 transmission requires thinking in terms of time of exposure rather than in terms of proximal distance.

49

50

51 **Keywords:**

52 *Indoor ventilation; COVID-19; airborne transmission; infectious risk assessment; health*
53 *policies; adequacy and respect of standards*

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57 **Highlights**

- 58 • *Relative airborne risk assessment following variant viral load and contagiousness*
- 59 • *Indoor analytical risk assessment including absence of ventilation*
- 60 • *Adequacy of the present norms of ventilation to Covid-19 pandemic*
- 61 • *Observation of non-compliance to standards concerning CO₂ Indoor Air Quality*

62 1 Introduction

63 Since its emergence at the end of 2019 a variety of public and health measures and
64 recommendations have been decided in several countries to contain COVID-19 spreading.
65 Recommendations pertain more to personal hygiene as, for example, washing hands, coughing in his
66 elbow, and keeping a social distancing with other individuals. However, collective measures have
67 been often more coercive. They include, amongst others, lockdown, closing of specific activities such
68 as restaurant services, quarantine, sanitary pass and last but not least human surveillance data
69 tracking. These mitigation measures have often had profound side effects, sometimes deleterious, on
70 the economy and population mental health [1].

71 Developing a rational basis for prevention is necessary to avoid irrational measures such as
72 forbidding outdoor activity in under-crowded area or organizing a kind of carousel circulation in
73 commercial centers. This requires identification of causal mechanisms, i.e. risk factors, explaining the
74 spread of the disease. A rational public health policy requires careful evaluation of the
75 pharmaceutical and non-pharmaceutical interventions. This should be the key role of epidemiology
76 [2].

77 As described in a large number of publications, there are three routes of transmission of
78 respiratory diseases. The first can be considered as a person-to-person transmission, occurring via
79 direct close contact, when microdroplets of physiological fluids emitted by an infected person are
80 projected directly on the mucosa (lips, nose, eyes) of another person in a kind of ballistic way. The
81 second one is linked to self-touch of the face mucosa by hands contaminated by surfaces ((fomites)
82 or projections. The third route, known as “aerosol” or “airborne”, is due to the creation of a
83 persistent aerosol of microdroplets in a range of size which prevents their rapid sedimentation on
84 the floor. This aerosol emitted by an infected person can be re-breathed leading to further
85 contamination. Mainly due to historical reasons [3] it was outright denied by most of health
86 authorities including WHO, or governmental agencies such as the CDC in the US (Center of Disease
87 Control) or the HAS (Haute Autorité de Santé) in France. Then mitigation measures were decided
88 considering the first two ways of transmission: social distancing, washing hands etc. Unfortunately
89 for public health, the consideration of airborne transmission should have led to a variety of other
90 decisions, especially in the field of indoor air quality (hereafter IAQ).

91 Ironically, knowledge was available for suspecting the importance of airborne transmission in
92 the COVID-19 pandemic. As soon as the first half of the last century, Wells and his co-workers have
93 led numerous experiments and developed concepts still largely in use nowadays in the field of
94 respiratory diseases. Wells has exposed his visionary ideas and summarized his work in a book of
95 1955 that any epidemiologist should have read [4]. With his coworker Riley he developed the famous
96 Wells-Riley model [5] which has been the basis of a lot of avatars and developments, especially in the
97 last two decades [6,7].

98 The non-consideration of airborne transmission has led L. Morawska, a leading scientist in the
99 field, to raise an alarm on its importance [8], followed by a call co-signed by more than two hundred
100 researchers in the mainstream press [9]. Nowadays the very importance of airborne transmission of
101 the COVID-19 disease is largely recognized and the reader is referred to the review in Science (and
102 references therein) of Wang et al [10], leading to the conclusion that airborne transmission is the
103 major spreading route. Complementary details can be found in [11-16].

104 Viruses mutate constantly, leading to new variants, eventually more infectious than the
105 previous strains, modifying the epidemiology of the disease. Variant classification is beyond the
106 scope of the present paper and rather complicated since there is not a single nomenclature. Their
107 scientific name refers to their lineage (a lineage is a group of closely related viruses with a common
108 ancestor) and to mutations resulting from changes in the genetic code leading eventually to new

109 variants [17]. An expert group of WHO has recommended using letters of Greek alphabet to name
110 variants in non-specialized audience [18]. Recently it was shown that the δ variant (B.1.617.2), which
111 appeared first in India in October 2020, leads to a much higher viral load (hereafter VL) in respiratory
112 fluids than initial strains, referred hereafter as IS [19-21]. According to recent observations, the new
113 omicron variant, spreading very fast in a number of countries, has a smaller VL than the δ one but is
114 nevertheless more contagious for microbiological reasons.

115 We rationalize below why new variants lead to a much higher airborne transmission,
116 essentially for the case of homogeneous transmission in indoor environment, following the Wells-
117 Riley approach. The relative risk for different variants (following VL and microbiological
118 characteristics), and various situations, is calculated. We have also performed measurements of CO₂
119 concentrations in a variety of environments, demonstrating that in the real-life ventilation is
120 seriously insufficient and that the homogeneous hypothesis is most often verified. We finish by
121 emphasizing the implications for health policy of the increased airborne transmission, which is
122 certainly the main transmission way for new variants. Following other authors [22] we insist on the
123 importance of **the time of exposure** although unfortunately most of the public policy is based on the
124 **distance of exposure**, probably due to the initial denial of airborne transmission.

125

126 2 basic notions and models in airborne transmission

127 2.1 Infectious particles and VL.

128 Particles emitted by a human refer either to spherical microdroplets or to more or less
129 hydrated “dry nuclei”, resulting from water evaporation of the respiratory fluids, which, beside
130 water, contains minor components like mucus, proteins and viruses [23]. VL is a key parameter of
131 particle infective power and depends on the mean number of viruses per unit volume of respiratory
132 fluids, which lead to a mean number per particle. This latter is statistical, i.e. it implies a large
133 distribution of particles with various viral contents. A mean VL per particle lower than unity implies
134 that some microparticles will contain a virus and others will not. Moreover, evaporation of exhaled
135 microdroplets can result in particles of lower size without virus loss. Since the smallest particles are
136 very abundant, they can be very efficient in airborne transmission.

137 These particles can be characterized by their size and composition, including VL which depends
138 on the viral strain. Their size depends mainly on their origin from the respiratory tract and of their
139 evolution in the ambient air, including evaporation. The largest droplets, behaving in a ballistic way,
140 are most often emitted by talking, sneezing, or coughing. The smallest ones come from various parts
141 of the respiratory tract, including the lungs. They have a large distribution of sizes, and many are
142 below 10 μm , especially after evaporation of some of the largest ones. In a kind of reversible way,
143 the smallest ones ($< 5 \mu\text{m}$) can penetrate deep in the lung when re-breathed and are known as
144 respirable aerosols [10,24].

145 One of the most sophisticated apparatuses used for the size characterization of these aerosols
146 is the specific wind tunnel developed by L. Morawska and her coworkers at the Queensland
147 University of Technology, at Brisbane, in Australia. It uses a variety of sizing techniques [25,26]. They
148 found four main modes in the distribution of particle size, centered around 0.8, 1.8, 3.5, and 5.5 μm
149 respectively.

150 2.2 Concepts of dose and quantum

151 As discussed in Rowe *et al.* [6] and others [27,28] the notions of level and dose of exposure
152 are easily defined for chemical or physical hazards (such as toxic gases or asbestos): the level of

153 exposure is then the concentration of toxic and the dose the quantity inhaled, ingested etc. These
 154 definitions are much more difficult for biological pathogen agents that are not easy to measure and
 155 have the possibility to replicate in the target host [28,29]. Concerning aerosols and as stated by Haas
 156 *et al.* [28] “precise information on the concentration of pathogens in aerosols has a lot of uncertainty
 157 associated with it”. Moreover, and for any kind of disease (i.e. respiratory, digestive etc.), the effect
 158 of the dose could depend on the way of transmission: inoculation, ingestion, airborne etc. Having
 159 defined a dose, the work of epidemiology is to assess quantitatively the risk for a given dose: by
 160 nature, such an assessment is statistical; it results most often in a law linking the probability of
 161 infection to the dose.

162 For airborne transmission of respiratory diseases, the definition of a dose is far from being
 163 straightforward since measuring pathogen concentrations in the air is extremely difficult [28].
 164 Therefore, Wells [4] **defined the quantum of contagium** as a hypothetical quantity that has been
 165 inhaled per susceptible individuals (men or animals) when 63.2% (correspondingly to a Poisson dose-
 166 response law, see sub-section 2.4) of these individuals display symptoms of infection. Quantum is
 167 used throughout the present paper and contrary to what has been sometimes claimed [30], it has no
 168 dimension **but is a counting unit** (like moles compared to molecules). It considers a variety of
 169 mechanisms: inhalation of airborne particles, pathogen inhibition by host defenses (see
 170 supplementary materials¹, hereafter SM1-7) or other losses, before any replication will start in an
 171 infected cell. Therefore, it corresponds statistically to a number of pathogens higher than one.

172 However, these statistical concepts do not mean that very few pathogens are never enough to
 173 start infection, as assumed sometimes. Indeed, the so-called “single hit” models make statistical risk
 174 assessment considering a very small probability, although non-zero, of infection by a single pathogen
 175 [28,31-34]. Further, and as stated by Haas *et al.* [28], the term of Minimum Infective Dose is very
 176 misleading since “Minimum” suggests some threshold effect for the infection. They emphasize that it
 177 corresponds in fact to the average dose administered and most frequently relates to the value
 178 required to cause half of the subjects to experience a response; they suggest that “median infectious
 179 dose” should be more appropriate, and they show that it is not possible to infer the probability of
 180 infection by a single pathogen from the magnitude of the median infectious dose.

181 2.3 Link between the quantum production rate and infectious aerosols

182 Evaluation of quantum concentration in air requires knowing the production rate of quanta by
 183 an infector, defined per unit time (unit: h⁻¹ for example). It can be deduced from epidemiological
 184 observations [35] but also linked to the distributions of microdroplets emitted by humans, together
 185 with the knowledge of VL in respiratory fluids and of the efficiency of the viral strain.

186 Following Buonanno *et al.* [36] the production rate of quanta q can be written as:

$$187 \quad (1) \quad q = VL \times c \times p \times \int_0^{10\mu m} N_d(D) \times dV_d(D)$$

188 where VL refers to unit volume viral load of respiratory fluid, c is a proportionality factor between
 189 the exhaled viral content (copies/unit time) and quanta, p is the pulmonary exhaled volume rate
 190 (volume/unit time), $N_d(D)$ the size distribution of droplet concentration (diameter D) of volume V_d .
 191 The factor c depends on the microbiological characteristics of the variant and can explain a higher
 192 value of q (and hence a higher contagiousness) even with a lower VL .

193 Equation (1) implies that **the production rate of quanta can be considered as proportional to**
 194 **VL in the respiratory fluids and to a factor (c) which depends on the virus microbiological**
 195 **characteristics**. Equation (1) assumes a single mean value of VL. This is a reasonable assumption
 196 since the quantum production rate is a statistical mean quantity that does not consider the diversity
 197 of particle emission processes, although VL depends probably on the particle origin from the

198 respiratory tract. Note also that the integral in (1) is just the volume fraction of emitted
199 microdroplets.

200 2.4 Dose calculation and infection probability

201 In absence of masks the dose of inhaled quanta can be expressed as the integral over time of
202 exposure of the product of quantum concentration n_q (quanta per unit volume) by the pulmonary
203 volume inhalation rate p (volume per unit time):

$$204 \quad (2) \quad X = \int_0^t n_q \times p \times dt$$

205 Note that this definition does not require a homogeneous distribution of quanta in space. Only
206 $n_q(\vec{r}, t)$ at mouth and nostrils location has to be considered. Also due to the extremely low
207 concentration of quanta in air, $n_q(\vec{r}, t)$ is not really continuous but can be treated as such due to the
208 statistical aspect of the problem (as discussed previously for the VL of microdroplets).

209 This dose X has no dimension but is dependent of the choice of the counting unit with its
210 dose-response (probability) function, which, for quanta, is the Poisson law [5]:

$$211 \quad (3) \quad P = 1 - \exp(-X)$$

212 For $X \ll 1$, this probability of transmission is then just X .

213 There are several other dose-response functions and dose definitions that can be used
214 [27,28,37]. In any cases, the probability of infection must be a monotonically increasing function of
215 the dose, starting from zero at dose zero and increasing toward an asymptote $P = 1$ at large dose.

216 2.5 models of transmission.

217 Whatever the chosen counting unit for the pathogens (viruses, quanta, particles), dose
218 evaluation requires to determine spatio-temporal evolution of their concentrations. For quanta it is
219 possible to distinguish between homogeneous models for which:

$$220 \quad (4) \quad \frac{\partial n_q(\vec{r}, t)}{\partial \vec{r}} = 0$$

221 and inhomogeneous ones which consider the possible gradients of n_q in space:

$$222 \quad (5) \quad \frac{\partial n_q(\vec{r}, t)}{\partial \vec{r}} \neq 0$$

223 In both cases the determination of n_q evolution uses conservation equations, described in
224 SM2, together with the well-mixed room hypothesis employed in homogeneous models.

225 The temporal evolution of quantum concentration in the homogeneous case reads (see SM2):

$$226 \quad (6) \quad n_q(t) = n_q^\infty \times \left[1 - \exp\left(-\frac{t}{\tau_1}\right) \right]$$

227 with:

$$228 \quad (7) \quad \tau_1 = \frac{V}{q_2 + \frac{V}{\tau_i}}$$

229 V being the room volume, q_2 the room ventilation rate and τ_i the virus lifetime.

230 The concentration of quanta for a number of I infectors, at stationary state *i.e.* for $t \sim$ a few τ_1 is:

231 (8)
$$n_q^\infty = \frac{I \times q}{(q_2 + \frac{V}{\tau_i})}$$

232 which, if the virus lifetime is long enough, reduces to:

233 (9)
$$n_q^\infty = \frac{I \times q}{q_2}$$

234 Note that if there is some air treatment (filtration or sterilization or both) for the volume V , it
 235 can be considered as an increase in the flow rate of fresh air and therefore results in an increase of
 236 q_2 value. Indeed, it is also possible to introduce the virus lifetime as an increase in the ventilation
 237 flow rate through equations (7) and (8). The virus lifetime τ_i depends on a variety of phenomena
 238 including UV irradiation.

239 In a situation where the stationary state has already been reached in a homogeneous volume
 240 at the beginning of exposure then, following equation (9 and 2), the inhaled dose is:

241 (10)
$$X = \frac{I \times q \times p \times t}{q_2}$$

242 which yields for the probability of transmission:

243 (11)
$$P = 1 - \exp\left(-\frac{I \times q \times p \times t}{q_2}\right)$$

244 Together with the quantum definition, these equations are the basis of the Wells-Riley model
 245 [5].

246 Note that conservation equation (see SM2) allows to consider any unsteady cases, including
 247 the case of very poorly ventilated rooms which is equivalent to $q_2 \ll V/t$, t being the time of
 248 exposure. Then, assuming a zero quantum concentration at $t = 0$ (case of a tutorial room at the
 249 beginning of a lecture after a weekend for example) the dose of exposure now reads:

250 (12)
$$X = \frac{p \times I \times q \times t^2}{2 \times V}$$

251 which is valid at $t \ll \frac{V}{q_2}$ and can be used with the Poisson probability law.

252 In many circumstances homogeneous models are completely relevant to indoor situations, as shown
 253 by measurement of CO₂ used as an indicator, or by considering turbulent indoor flow with typical
 254 velocities around 0.1-0.2 m/s induced by natural or mechanical ventilation or by air movement due
 255 to plumes from occupants or any hot surface. However, there are undoubtedly conditions where
 256 substantial gradients of pathogens (quantum) prevail leading to a risk which is dependent on the
 257 indoor position of infectors and susceptible persons. Two situations can be depicted for
 258 inhomogeneous transmission: the case of indoor viral transport on rather large distances, i.e., which
 259 are close to the space typical length [22] and the event of close contact between an infector and a
 260 susceptible person [37]. The concepts described above for homogeneous models are still valid but
 261 now the determination of $n_q(\vec{r}, t)$ requires solving transport equations as described in SM2. Note
 262 that It is now largely admitted that the transmission of COVID-19 disease by close contact is most
 263 often an airborne one, referred in the literature as "short-range airborne transmission". In their
 264 paper, Cortellessa et al. [37] have also considered large microdroplets which are assumed larger than
 265 100 μm in diameter in their model. Beyond this size the authors supposed that microdroplets do not
 266 suffer evaporation and have ballistic trajectories whereas below 100 μm , they evaporate and
 267 consequently reduce in size in such a way that they can be airborne. From Cortellessa et al. analysis,

268 the large microdroplets ($> 100 \mu\text{m}$) prevail only at very short distance ($< 60 \text{ cm}$), with a contribution
 269 to the dose being completely negligible further.

270 This demonstrates the airborne character of most airborne contamination in close contact, excepted
 271 intimate. Other implications of this work are found in SM3.

272

273 **3 Relative risk assessment following variant VL and contagiousness**

274 3.1 General formulation.

275 As developed previously, airborne models of infection usually introduce a dose of exposure
 276 X to an infective agent, which is assumed proportional to VL in the respiratory fluids. Then the
 277 probability of infection follows a dose-response function.

278 **All other parameters being equal** (time of exposure, flow rate of fresh air etc..), it is then
 279 possible to assess a relative risk between two variants (in a way similar to Rowe *et al.* [6] for the
 280 relative outdoor versus indoor risk). For sake of simplicity, we concentrate the following discussion
 281 on the initial strain and the δ variant with different VL, VL_{IS} and VL_{δ} respectively.

282 Let R be the ratio of the doses of exposure between IS and δ in case of identical situations,
 283 from section 2 (Eq. 1 and 2), R can be reduced to the ratio of VLs and of the proportionality factors c :

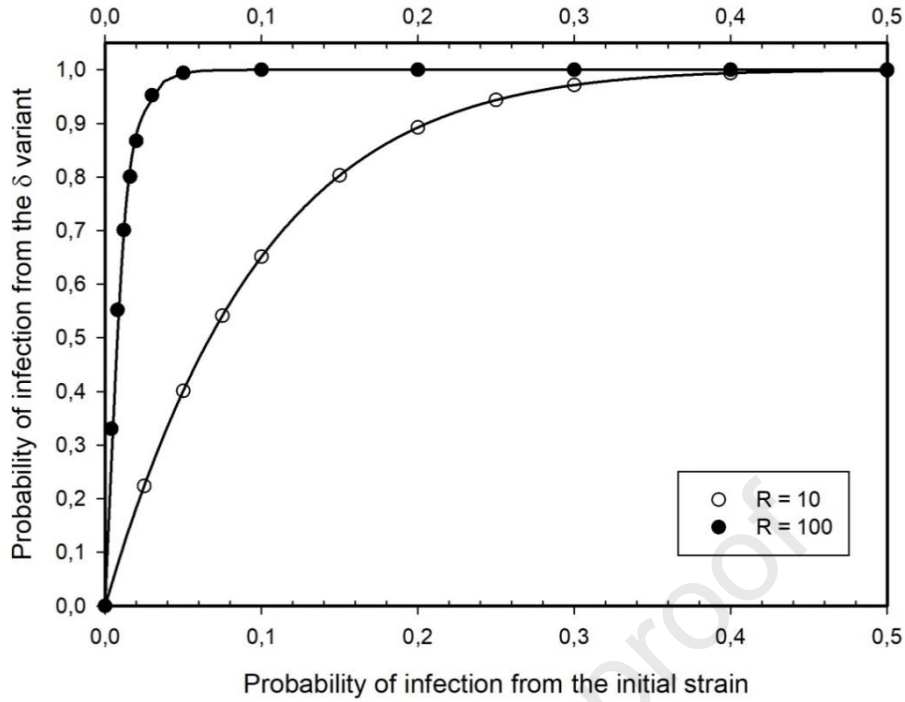
$$284 \quad (13) \quad R = \frac{X_{\delta}}{X_{IS}} = \frac{VL_{\delta}}{VL_{IS}} \times \frac{c_{\delta}}{c_{IS}}$$

285 It is then easy to demonstrate that relative probabilities of being infected between
 286 respectively δ and IS variants follow the next equation:

$$287 \quad (14) \quad P_{\delta} = 1 - (1 - P_{IS})^R$$

288 which for $P_{IS} \ll 1$ reduces to $P_{\delta} = R \times P_{IS}$.

289 It results that, from the recognized fact that $VL_{\delta} \gg VL_{IS}$, the airborne contamination by the δ
 290 variant is much more efficient than with initial strains for comparable situations, as shown in Figure 1
 291 for $R = 10$ and 100 respectively. Note that the same conclusion could apply with the omicron variant
 292 (the subscript δ should be replaced by o) but then the c_o factor would also explain the higher
 293 contagiousness.



294

295 Figure 1: δ probability of airborne infection versus initial strain for a ratio of VL in respiratory fluids
 296 of 10 and 100 (all other parameters being equal).

297 3.2 The case of public access area.

298 We will examine first the case of an indoor space ventilated following the norm and at
 299 stationary state. Then, the dose of exposure is given by equation (10), and, in the Wells-Riley model,
 300 the probability of infection follows the Poisson law (11). If the ventilation of the public space
 301 q_2 conforms to the norm per person q_{norm} :

$$302 \quad (15) \quad q_2 = q_{norm} \times N_p$$

303 with N_p being the number of persons within the area. This assumption is of course questionable
 304 either if this norm is not followed or if the value of q_2 is fixed constant, independently of N_p as it is
 305 often the case.

306 Assuming an infector proportion r , we can express the number of infectors as:

$$307 \quad (16) \quad I = r \times N_p$$

308 Strictly speaking it is the prevalence of infectors, including asymptomatic, that should be used
 309 for r . It is anyway probable that the number of infectors is proportional to N_p . As discussed in SM4 it
 310 is extremely difficult to have the exact value of r from the values of positivity rate or incidence rate
 311 reported by health agencies. Below we use a "reasonable" value for r consistent with the pandemic
 312 situation in Brittany in November and December 2021 during our series of measurements detailed in
 313 the next section 4.

314 the dose of exposure results:

$$315 \quad (17) \quad X = \frac{r \times p}{q_{norm}} \times q \times t$$

316 which clearly shows the multifactorial character of the risk. In the case where the ventilation
 317 conforms to the norm and for a given value of r , the difference between a school, a restaurant and a

318 commercial center comes essentially from the time of exposure t . Note that this time is a total time
 319 which does not need to be continuous but can be a summation of hourly and daily exposition in the
 320 various spaces that the individual went through, due to the fact that the risk is essentially
 321 probabilistic. **Clearly the difference in quantum production rate between δ variant and previous**
 322 **strain, plays an enormous role** in the dose, and hence in the probability of infection. However, it is
 323 clear from equations (11) and (17) that the known parameters on which it is possible to play are the
 324 time of exposure t , the ventilation rate q_2 itself, depending on the norm of ventilation q_{norm} and on
 325 the number of persons in the volume, if the total ventilation conforms to the norm.

326 Note that when N_p is not very high, Eq. (16) may lead to a number of infectors $I, I < 1$, which
 327 could seem unrealistic. Instead of the use of Eq. (17) for the dose used with the Poisson probability
 328 (hereafter P_{WR} – Eq. (11)) the following value of the probability should be used:

$$329 \quad (18) \quad P = \sum_1^{N_p} P_n(r) \times P_{WR}(n)$$

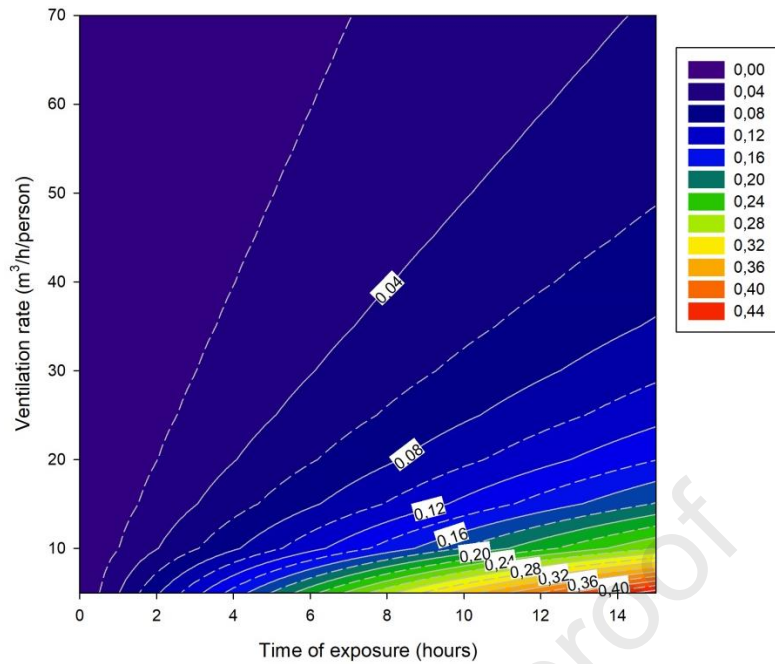
330 where $P_n(r)$ is the probability to have n infectors and $P_{WR}(n)$ the probability of being infected with n
 331 infectors.

332 Then, it can be shown, (see SM4) that equations (3) with (17) lead to a very similar result than
 333 the more exact calculation (18), assuming that the ventilation rate follows equation (15).

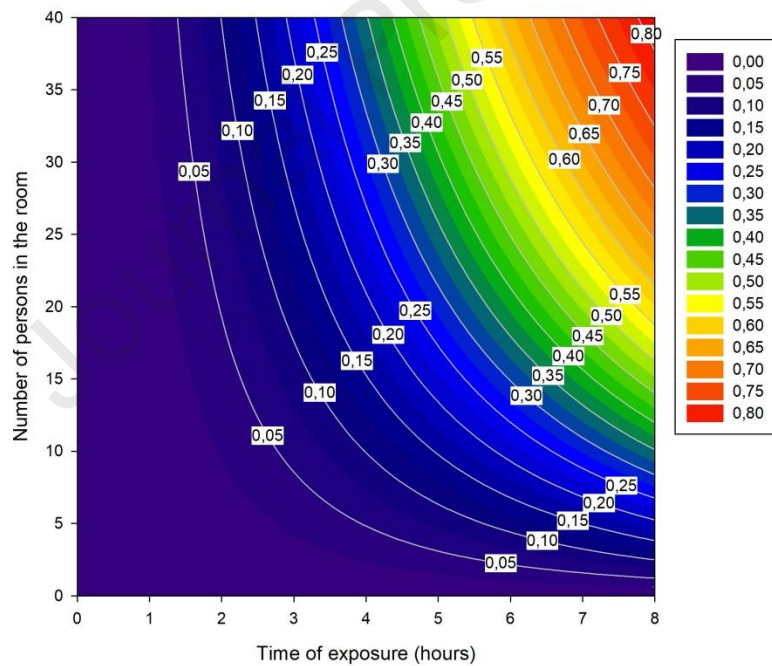
334 In Figure 2, the curves of equal probability of infection versus the time of exposure and the
 335 ventilation volumetric flow rate (starting at 5 m³/h/person) are shown, for a quantum production
 336 rate of 40 h⁻¹, and an infector proportion $r = 0.01$. It is important to stress that quantum production
 337 rates found in the literature are very dispersed. For instance, in table 3 from Mikszewski et al. [38], a
 338 series of real cases are gathered with quantum production rates ranging from 15 to 4213 quanta per
 339 hour depending on the situation. Our choice aims at being somewhat representative although as
 340 commented in section 4, the influence of changing the present chosen parameters (within a
 341 reasonable range) on the probability calculation can be easily analyzed. Of course, in the real life, if
 342 the ventilation rate is fixed at the maximum space occupancy and not by equation (15) it would
 343 result in a smaller probability of infection in a non-fully occupied room. Note that this figure results
 344 from the assumption that the ventilation rate is proportional to the number of people in the well-
 345 mixed space.

346 In the case of very poor ventilation, we can use Eqs. (3 and 12) in order to estimate the risk in a
 347 public space as a function of the number of persons in the volume V and of the time of exposure,
 348 assuming that at time $t = 0$ the concentration of quantum is zero. This could be for example the case
 349 of a poorly ventilated tutorial room (i.e. $q_2 \ll \frac{V}{t}$) where the lecture (and hence the student
 350 presence) starts at $t = 0$; t being the time of exposure. Figure 3 displays the curves of equal
 351 probability of infection versus the time of exposure and the number of persons for an infector
 352 proportion of 0.01 and a volume of 150 m³.

353 Note that the wearing of masks will of course alter these figures by reducing the quantum
 354 production rate as well as the quantum inhaled quantity (see SM5).



355
 356 Figure 2: Probability of infection contours as a function of time of exposure and ventilation rate per
 357 person assuming a quantum rate of 40 h^{-1} , an expiratory rate of $0.50 \text{ m}^3/\text{h}$ and an infector proportion
 358 of 0.01.



359
 360 Figure 3: Probability of infection when the ventilation is poor (see II.5). Calculations are made using
 361 an expiratory rate of $0.50 \text{ m}^3/\text{h}$; a quantum rate of 40 h^{-1} ; an infector proportion of 0.01 and a room
 362 volume of 150 m^3 typical of a lecture room.

363

364 **4 Analysis of some specific cases**

365 4.1 Observations

366 As discussed earlier, aerosols are the main contamination routes of COVID-19 and exposure
 367 becomes critical indoors. It is now widely admitted that ventilation is, beside the mask, the most

368 effective way for reducing indoor airborne transmission [8,13,39,40] in particular for highly insulated
 369 and airtight buildings, where the building envelop infiltration is reduced to a minimum to respect
 370 thermic regulation. The measure of indoor CO₂ concentration is considered in standards as an
 371 indirect measure of IAQ [7] or as a proxy of ventilation rate. One should distinguish the indoor CO₂
 372 limit values (1000 to 1300 ppm) issued from building ventilation regulations [41,42] from maxima
 373 recommended in the current sanitary context: 800 ppm wearing a mask and 600 ppm without a mask
 374 [43,44]. In fact, as recalled by Li [40], outside of healthcare settings, existing ventilation standards do
 375 not account for infection control. When CO₂ concentration exceeds threshold values, the ventilation
 376 flow rates are usually insufficient and aerosol route contamination risk is high as illustrated by
 377 Figures 2 and 3.

378 In this context, we carried out, in autumn 2021, a series of CO₂ concentration measurements
 379 and observations in various environments. Measurements consisted in determining the CO₂ time
 380 evolution within each room using non-dispersive infrared (NDIR) CO₂ sensors (Aranet 4 or ZG-106
 381 Protronix CO₂ monitor). Their accuracy was ±3% and ±5% of reading for the Aranet 4 and the ZG-106
 382 Protronix respectively. The sensors are factory-calibrated and allow raw data logging with time
 383 stamps. Sensors were positioned between 1 and 2 m height (corresponding to the occupants head
 384 position), at least 2m far from every person and distant from windows or doors.

385 Further, when possible the mechanical ventilation was directly measured by using a balometer
 386 from ACIN (Flowfinder mk2). The accuracy in flow rate measurements was ±3% of the reading. Three
 387 categories of spaces were investigated including two university lecture rooms (ULR5 and ULR-20) and
 388 one pupil schoolroom; two university amphitheatres (UAW and UAE) and finally a restaurant. For
 389 each room, the main characteristics are given in Table 1. This includes, among others, the maximum
 390 allowed people from which the regulatory ventilation is determined according to French regulation
 391 [42] which specifies the flow rate per person (PFR hereafter) as being 18 m³/h/person for lecture
 392 rooms and amphitheatres; 15m³/h/person for the schoolroom and 22 m³/h/person for the
 393 restaurant. A time step of 10 minutes was sometimes fixed in accordance with the French IAQ decree
 394 n° 2012-14 [45] for five-days monitoring to determine the ICONE index (see SM6).

395 The CO₂ time evolution followed the standard law:

$$396 \quad (19) \quad [CO_2] - [CO_2]_0 = ([CO_2]_\infty - [CO_2]_0) \left\{ 1 - \exp\left(-\frac{Qt}{V}\right) \right\}$$

397 where [CO₂]₀ is the CO₂ concentration, expressed in ppm, at the beginning of the analytical fit ($t = 0$),
 398 [CO₂]_∞ is the stationary CO₂ concentration ($t = \infty$), Q the ventilation flow rate (m³/h), V the room
 399 volume and t the time at which the measurement was carried out. From this equation, it is
 400 straightforward to determine the ventilation flow rate Q from an exponential fit of the measurement
 401 when the volume V is known, at least when [CO₂]_∞ is not ill-defined, a situation that occurs when the
 402 number of people constantly changes with time like in the restaurant (see Table 1).

403 The CO₂ time evolutions are illustrated in Figure 4-(a-d) where the reference of the CO₂
 404 concentration has been taken as an outdoor [CO₂]_{ext} usual value of 400 ppm instead of considering
 405 [CO₂]₀ as the reference. This makes it easier for the readers to return to the absolute value since the
 406 initial [CO₂]₀ is never the same from one test to another.

407

408

409

410 Table 1: Ventilation measurements for various environments with their own main characteristics

Room	ULR5	ULR20	Schoolroom	UAW	UAE	Restaurant
Volume (m ³)	136	402	173	900	1035	--
People/max	28/30	67/68	30/30	40/142	95/163	var./120
measurement duration/time step (min)	80/10	90/5	7days/10	56/ var.	55/1	5days/5
Ventilation system ^a	U	B-dyn	H	B	B	B or B-dyn
Regulatory volumetric flow rate (m ³ /h)	540	1224	450	2556	2934	2640
Volumetric flow rate from CO ₂ (m ³ /h)	53	1124/450	50-100	2576	1219	--
Measured volumetric flow rate (m ³ /h)	--	Max/Min = 1187/200	--	--	1009	~ 500

411 ^a B: bidirectional ventilation; B-dyn: bidirectional dynamic ventilation; U: unidirectional ventilation; H:
412 hybrid ventilation

413 Figure 4-a compares two lecture rooms (ULR5 and ULR20, see Table 1). These lecture rooms
414 are at a University building over 50 years old, which has not yet undergone any energy retrofit. The
415 ULR5 is equipped with air intake vents installed in window frames. As the building envelope is not
416 airtight and since the toilets facilities, equipped with mechanical air exhaust, are far away from ULR5,
417 little fresh air enters by the windows intake vents. In addition, exhaust flow rates at the level of the
418 building are too low compared to the regulatory ventilation needs. This explains the observed very
419 poor IAQ with maximum concentrations of CO₂ exceeding 5000 ppm. This trend has been confirmed
420 in a similar lecture room (ULR4, not shown for brevity) where CO₂ concentration measurements
421 during five consecutive scholar days lead to an air stuffiness index ICONE of 4, i.e. very high
422 confinement (see SM6).

423 The ULR20 is a lecture room, among three rooms of the same previous building, which were
424 fitted more than ten years ago with a common dynamic two-way ventilation system, using the level
425 of CO₂ in the exhaust circuit to control the ventilation flow rate. This system sized for a maximum
426 flow rate of 1187 m³/h (for occupancy capacity of 68 students plus a teacher, i.e. 17.2 m³/h/person
427 close to the French regulatory value of 18 m³/h/person). It is however set at a minimum flow of 200
428 m³/h during the unoccupied hours, and is manually switched off during holidays. In this room, on
429 2022/01/03, while the ventilation was still off after holidays, a maximum concentration of 3300 ppm
430 was registered after one hour during an exam gathering 64 persons. The corresponding evolution is
431 not given for brevity. During normal operation of the ventilation system of full occupied ULR20, the

432 CO₂ level does not exceed 1700 ppm (see Figure 4-a). This threshold corresponds to a Category 3
433 classification (moderate level may be used for existing buildings) in the UE regulation [43,46] and is
434 above the French limit value of 1300 ppm [42]. However, this remains acceptable in comparison with
435 the previous ULR5 case.

436 Figure 4-b presents CO₂ evolutions in two lecture halls (UAE and UAW). UAE is, as previously,
437 over 50 years old, whereas UAW is inside a modern new building. One can observe that in UAE the
438 CO₂ concentration reaches a high value of 2100 ppm after a one hour lecture gathering 95 persons.
439 This corresponds to a PFR of 13 m³/h/person. Note however that when the lecture hall is full, the PFR
440 would then be equal to 8 m³/h/pers., which is very far from the regulatory value. On the opposite,
441 UAW seems very well ventilated since the CO₂ concentration did not exceed 600 ppm in the presence
442 of 40 persons. The deduced volumetric flow rate was as high as 2576 m³/h, which results in PFR = 18
443 m³/h/person when considering the UAW maximum capacity of 142. Therefore, this lecture hall
444 complies with French regulations, and probably when it is full, the CO₂ would be in the regulatory
445 range 1000 to 1300 ppm [42]. However, we can regret, **for energy consumption reasons**, the
446 apparent absence of flow rate control as a function of the occupancy density.

447 Figure 4-c shows the CO₂ time evolution acquired during one full week in a classroom. The
448 building is old (built almost a century ago) and has not benefited from any energy retrofit. The
449 considered schoolroom receives 30 pupils 7-years-old. The insert in Figure 4-c gives an example of a
450 CO₂ rise from which the ventilation rate could be estimated. Since, the flow rate was found quite
451 small, the measurements presented some dispersion from one day to the other but the observed
452 range (50-100 m³/h) is very far below the regulatory flow rate for a schoolroom with a maximum
453 occupancy of 30 persons (i.e. 450 m³/h according to the French regulation [42,47]). The
454 corresponding air stuffiness index [45] is ICONE=4, corresponding to very confined class. This
455 observation joins those of the French IAQ observatory [48] and various literature studies of
456 ventilation state in schools in France [49,50] and elsewhere, particularly in Europe or USA [51]. In this
457 latter investigation, Fisk performed a thorough review, which demonstrated the widespread failure
458 of ventilation systems to provide the minimum flow rates specified in standards for classrooms. He
459 reported that the maximum peak CO₂ concentrations ranged from about 3000 to 6000 ppm. It is also
460 important to stress that the French standard [42,47] makes the differentiation between young
461 children (under 15 years old, PFR = 15 m³/h/person) and older teenagers or adults (older than 15
462 years, PFR = 18 m³/h/person), whereas this is not biologically relevant [52] because young children
463 emit as much CO₂ as older ones or adults. Children being more fragile than adults, the individual PFR
464 should on the contrary be higher for them. The UE Regulation [43] recommends a PFR = 36
465 m³/h/person in the best IAQ category (category 1) for sensitive and fragile persons with special
466 requirements, which should be the case for young pupils.

467 Finally, we carried out a CO₂ monitoring during a week (Figure 4-d) in a modern restaurant
468 situated in a coastal location of the Department of "Côtes d'Armor" in France. We used two Aranet
469 sensors each one set in one of the two lunchrooms of the restaurant which communicate to each
470 other through a large aperture. The two sensors were approximatively at a distance of 10 m to each
471 other and demonstrate a similar CO₂ concentration along the week. This is a strong demonstration
472 that for this case, the well-mixed assumption holds. Interestingly, the restaurant is exposed to the
473 wind, which can cause large variations in air renewal flow rates. Observations correlate strongly with
474 an enhancement of ventilation with the strength of the wind (and inversely for CO₂ concentration)
475 which is shown on each peak of the figure in Beaufort scale (Bt = 1-2 on Monday; 4-5 on Tuesday; 5-7
476 on Wednesday; 5-6 on Thursday and 5-7 on Friday). Not indicated is the direction of the wind which
477 has been changing continuously along the week. The high variability in peak CO₂ from day to day can

478 be clearly seen in Figure 4-d and wind effect on the level of airing appears obvious. On Monday,
 479 when there was no wind, a maximum concentration of 1800 ppm was recorded, which is a high level
 480 compared to the French public health committee recommendations to not exceeding 600 ppm in
 481 situations in which attendees are not wearing a mask [44].

482 Furthermore, in essence the restaurant is a place where conditions are continuously variable
 483 (customers do not arrive at the same time, doors open frequently) and it is not easy to establish
 484 stable conditions allowing to determine air flow rates from CO₂ concentrations. Moreover, even if we
 485 do not have the confirmation, it is very likely that the bidirectional ventilation is dynamic, which
 486 makes air flow rates variable. The in-situ volumetric flow rate measurements done in customers'
 487 space (lunchrooms and bar), lead to a total air flow rate around 500 m³/h. The hood in the kitchen
 488 and the related compensation grille, placed on opposite exterior wall, have probably an effect on
 489 flow patterns in lunchrooms, as the kitchen door is kept open during lunchtime. Since our objective
 490 was to evaluate the potential risk of contamination in a space where masks fall, we did not focus too
 491 much on a precise determination of the ventilation rate considering the above-mentioned
 492 difficulties. Rather we concentrated on the CO₂ levels achieved every day (see discussion in section
 493 4.2).

494 Through all above observations, the poor ventilation of the investigated premises is evident
 495 since most of our measurements range between one third and one tenth of the regulatory
 496 volumetric flow rates. Further to this failure in respecting norms, it is essential to understand that the
 497 present ventilation standards worldwide are not designed for infectious control, whatever the
 498 respiratory virus is. The present work also agrees with the large surveys of various bibliographical
 499 sources (Ribéron 2016, Canha 2016, Batiactu 2018) not only in France as revealed by the thorough
 500 review from Fisk [51]. Interestingly, in this latter study, Fisk mentions that increasing ventilation with
 501 annual costs ranging from a few dollars to ten dollars per person constitutes less than 0.1% of typical
 502 public spending on elementary and secondary education in the US. Such spending is judged a small
 503 price to pay given the evidence of health and performance benefits. This observation is more than
 504 ever true in this pandemic period and could be extended to other countries and other sectors than
 505 education. In the same spirit, it is desirable to generalize the use of CO₂ sensors, a very affordable
 506 tool, in buildings to assist people in applying the suitable mitigation behaviours such as windows
 507 opening for instance to accelerate indoor air renewal.

508 4.2 Risk assessment

509 For the various situations described above it is important to derive a risk probability for an
 510 exposed person (susceptible) as a function of the observed CO₂ concentration. From a statistical
 511 point of view and a large number of persons, the dose can be written (see SM7) as:

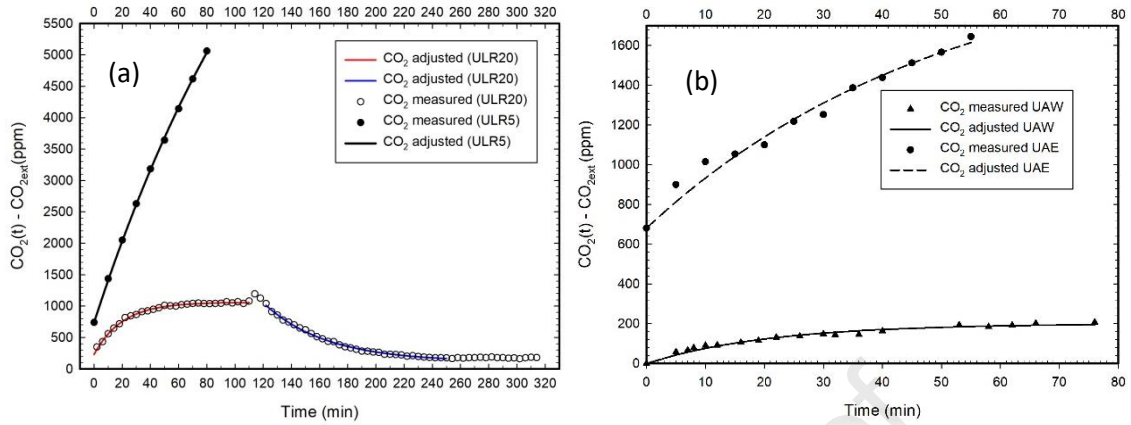
$$512 \quad (20) \quad X = \int_{t_0}^{t_1} \frac{\Delta CO_2(t)}{CO_{2,exh}} \times r \times q \times dt$$

513 This relationship is valid for any situation including environments with poor ventilations and
 514 transient situations as well as stationary states ($\frac{\partial \Delta CO_2}{\partial t} = 0$). It does not need the ventilation flow
 515 rate value. It can be extended to include a virus lifetime (omitted here for sake of simplicity), which
 516 does not change the conclusions. Parameters r and q are again the proportion of infectors and the
 517 quantum production rate respectively, $\Delta t = t_1 - t_0$ is the time of exposure of the susceptible,
 518 $CO_{2,exh}$ the quantity of CO₂ in the air exhaled by a human (~40000 ppm), ΔCO_2 the difference
 519 between the measured CO₂ in ppm and the outdoor natural level measured with sensors.

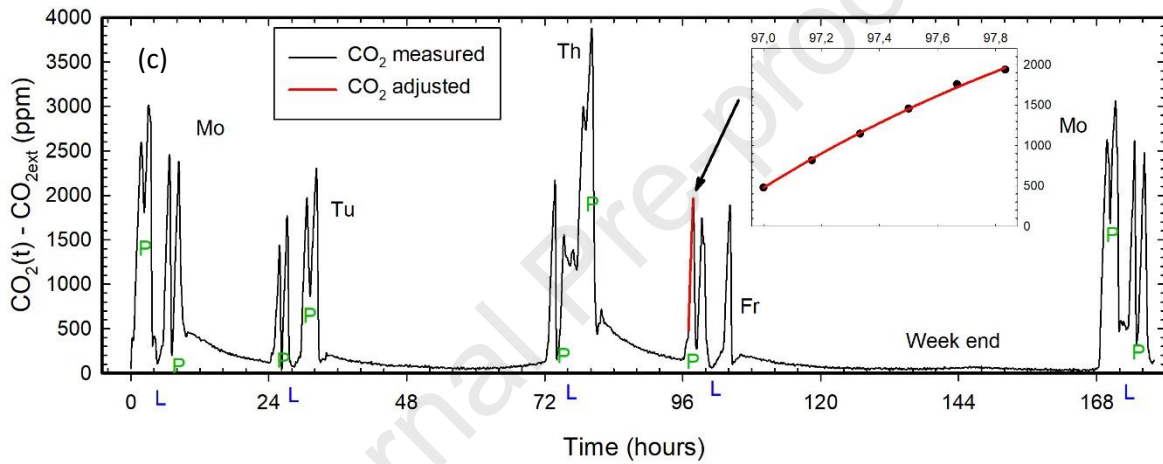
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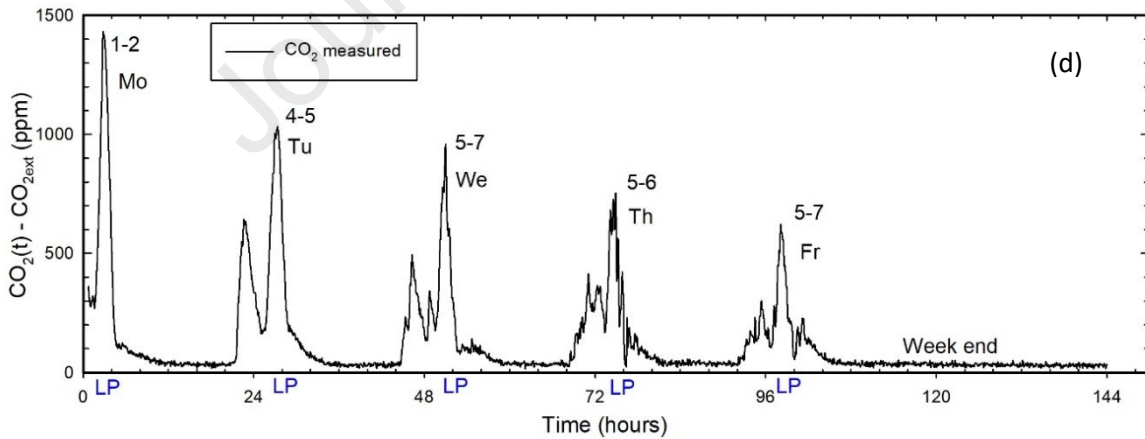
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526 Figure 4: CO₂ time evolution within examples of indoor spaces – complementary information are
 527 given in Table 1: (a) two lecture rooms (ULR5 and ULR20); (b) two lecture halls (UAE and UAW); (c)
 528 schoolroom over one week, L: Lunch, P: Playtime; (d) restaurant over a week (numbers close to the
 529 CO₂ peaks represent the strength of the wind in Beaufort scale, LP: Lunch Peak).

530

531

532

533 We can define a mean value of “human” CO₂ for the time of exposure Δt by:

$$534 \quad (21) \quad CO_{2,mean} = \frac{\int_{t_0}^{t_1} \Delta CO_2(t) \times dt}{\Delta t}$$

535 Then, the dose can be written:

$$536 \quad (22) \quad X = \frac{CO_{2,mean}}{CO_{2,exh}} \times r \times q \times \Delta t$$

537 which highlights, beside the CO₂ concentration, **the importance of the time of exposure Δt and of**
 538 **the number of infectors**. Note that the remarks made in section 3.2 for the r value remain valid. If a
 539 healthy subject is exposed to successive doses X_i corresponding to different periods of exposure Δt_i ,
 540 then the total dose is just the sum of the successive doses (cumulative risk):

$$541 \quad (23) \quad X = \sum_i X_i$$

542 Following these formulas, we can deduce some risk probabilities corresponding respectively to
 543 the situations described in section 4.1. They are summarized in Table 2:

544 Table 2: Probability of infection for various scenarios

	school		restaurant		UAE	UAW	URL5	URL20
$\Delta t(\text{min})$	1100		80	400	55	76	80	112
	No mask	mask	1 meal	5 meals	1 Lect	1 Lect.	1 Lect.	1 Lect.
P	0.237	0.027	0.005<X<0.012	0.040	0.013	0.001	0.040	0.013

545 In this table we have utilized the same values for r and q as for figures 2 and 3: 0.01 and 40 h⁻¹
 546 respectively. Mostly for the values of calculated doses, the probability for a healthy susceptible to be
 547 infected is just nearly equal to the dose, due to its low value as explained previously. Therefore, the
 548 influence of changing the values of r and q can be easily estimated by a proportional calculation, as
 549 long as the dose remains small.

550 Some points in this table merit to be highlighted:

- 551 • For the school, the situation would be catastrophic without the risk reduction due to the
 552 mask. However, the precise quantitative impact of mask wearing is difficult to evaluate
 553 as discussed in the SM5. Also the social acceptability of mask wearing by children merits
 554 to be discussed.
- 555 • For the restaurant/bar, we have considered that customers are mainly workers who
 556 spend about 80 minutes at lunch. The risk is negligible for a single meal. In Table 2 P is
 557 bracketed since conditions varied depending on the day. If the restaurant is visited on a
 558 daily basis (5 meals) risk could be raised to a few percent following equation 23.
 559 However, the calculation does not consider that the mask is partly worn in the
 560 restaurant. In any case our observation and calculation show that the risk here is not
 561 especially high, which questions public policy in this field.
- 562 • For the other premises, which are located at the university, observations show a
 563 considerable dispersion. The risk can be very high for a lecture room very poorly
 564 ventilated (case URL5 of table 2) as well as reasonable in well ventilated area (case
 565 UAW). It must also be considered that for the university premises we have not
 566 considered either mask wearing or the cumulative aspect of the dose. As discussed in

567 SM5, using masks induces a risk reduction of a factor of about 9. This is however easily
 568 counterbalanced within one week if students attend 9 lectures in the same room which is
 569 quite possible. It remains that in poorly ventilated areas the risk is high.

570

571 **5 Implications of increased airborne contamination for health policy**

572 The previous sections highlight the multiparameter character of the risk, through the time of
 573 exposure and the concentration of airborne infectious particles, itself linked to the proportion of
 574 infectors and to the indoor ventilation flow rate. With new variants such as δ or \omicron (omicron), the
 575 quantum emission rate q can be estimated orders of magnitude higher than with the original strain
 576 due to VL or microbiological characteristics. Then, the spread of the virus should be mainly airborne
 577 even for close contact, and much more efficient. This increased spreading is in fact observed [53]
 578 even if, fortunately, it seems that the new variants are much less lethal than the original Wuhan
 579 strain. Moreover, health policies have not been sufficient to slow down efficiently this new
 580 contamination, especially in Western Europe. With more dangerous variants or new respiratory
 581 diseases, either more lethal or more contagious or both, new intervention measures must be
 582 considered. In the δ or \omicron variant cases, the models and concepts presented in this paper and the
 583 experimental measures reported, lead us to derive implications **for health policy**. Such an exercise
 584 has already been done previously by leading scientists of the field [13] but it **seems that it has not**
 585 **been sufficiently considered** by health policies. Moreover, we do think that, beside a variety of
 586 engineering solutions already preconized by Morawska *et al.* [13] other mitigation measures are
 587 necessary, and we insist that authorities have to **change their mind in matter of priority**.

588 Amongst the various interventions of public policy discussed below we focus on the non-
 589 pharmaceutical ones. We first consider interventions directly targeting IAQ, i.e., mask, air filters and
 590 sterilizers, and ventilation. In this context we will also discuss the influence of the way of life, which
 591 depends on the country and the climate, and could lead to take immediate measures with strong
 592 positive consequences. We will then turn to interventions that are not directly targeting IAQ but
 593 nevertheless have implications on IAQ (e.g., living conditions during lockdown) or whose
 594 effectiveness is dependent of our understanding of contamination routes (e.g., contact tracing).

595 **Such discussion is all the more needed** that vaccine efficiency has been reported dropping far
 596 from 100% with time and variants for most vaccines, including Pfizer, and that their ability to stop
 597 transmission by asymptomatic infection is questionable [54,55]. Vaccination alone will not be enough
 598 to stop the epidemic spreading via airborne contamination, because present vaccines do not provide
 599 100% immunity, especially with new variants such as omicron, although they result in a strong
 600 reduction of illness gravity. Beside the need of a **large vaccination of people at risk** (elderly, diabetic,
 601 overweight etc.) to reduce disease severity, it is clear that mitigation measures especially toward the
 602 problem of IAQ, should be highlighted: checking of HVAC (Heating, Ventilation, Air Conditioning)
 603 systems, air monitoring or development of high flux air sterilizers. New variants or new respiratory
 604 viruses in the future require a change of paradigm in this field [56]. If measures implying
 605 technological developments can be implemented only on mid-term, measures directed toward
 606 people information and the way of life must be taken immediately.

607 5.1 Targeting IAQ

608 When IAQ is deficient, especially in indoor situation, **wearing a mask is certainly highly useful**
 609 [10,57,58], but their efficiency (especially for surgical ones) is not such that it could be the solution
 610 alone. It is possible to calculate that the risk probability P could be decreased by a factor of ten when

611 both infectors and susceptibles wear it (see SM5). However, with new variants the quantum
612 production rate increase could counterbalance this advantage. Moreover, in most countries, after a
613 deny of mask interest, the choice of surgical ones in the general population has been made, although
614 they are much less efficient than N95 respirators [59,60]. In some situations, the public should be
615 informed of the better choice, depending on the IAQ (see sub-section 4.2). People must be told that
616 wearing mask under the nostrils is inefficient.
617

618 Therefore we conclude that **wearing a mask alone, although useful, is insufficient to**
619 **counterbalance the very high VL due to delta variant or the microbiological characteristics of**
620 **omicron**. Also social acceptability of masks on the long term is most doubtful. Therefore, we must
621 take further corrective measures to improve IAQ.

622 IAQ has been recognized as a concern for public health and is addressed by building norms.
623 However, IAQ policy has mainly considered the issue of chemical and particulate matter pollutants,
624 excepted in the context of health care buildings, such as hospitals [61]. It is time to address the
625 question of airborne pathogens "pollution" in the general population and its consequences for
626 respiratory diseases. This will need a considerable change in the norms and recommendations for
627 buildings (Meslem *et al.*, in preparation), since, from this point of view, they are still in their infancy.

628 The problem is closely linked to building ventilation, which has been for centuries a natural
629 ventilation, i.e., fresh air intake by voluntary or involuntary leaks on the building envelope allowing
630 entrance and circulation of fresh air without real control. Since the first oil shock and the subsequent
631 implementation of increasingly restrictive energy regulations, including today new constraints linked
632 with environmental impact, things changed with buildings becoming more and more airtight and
633 with HVAC technologies allowing ventilation control. The admission of fresh air is therefore
634 minimized at the lowest value (hygienic flow rates) compatible with physicochemical IAQ, in order to
635 save energy but frequently this leads to non-compliance with regulatory hygienic flow rates.

636 We recommend, in the context of new buildings and retrofit that is put in place, the in-situ
637 **verification of regulatory flow rates**. This is often not done, because the regulations do not require
638 it, as it is the case in France in the context of the regulation RT2012 [62]. It follows, as exemplified in
639 this work, that **introduced fresh airflows are much lower than the regulatory values**. The ventilation
640 professionals published an alarming report on the failure of the ventilation systems and demanded in
641 2018 that a certificate of receipt of these systems be delivered, like the certificate of receipt of
642 airtightness of building envelopes mandatory in RT 2012 [63]. The next regulation RE2020 [64]
643 applicable since January 2022 for residential buildings, takes a step forward by setting up an
644 obligation to measure ventilation flow rates. However, one can object that this point is not subject to
645 a building acceptance certificate. Another criticism is that verification of the airflows is not entrusted
646 to an independent control office since ventilation system installers can make the flow rates
647 measurements themselves. The Swedish experience of the OVK (Obligatory Ventilation Control) in
648 place since 1991 [65] is shared in REHVA site [66] as an example to be followed by European
649 countries and elsewhere. The Swedish regulation specifies that the first inspection of the ventilation
650 system is mandatory when it is taken into operation. Then, regular inspections are mandatory every
651 3 or 6 years, depending on the building type (3 years interval for pre-schools, schools, and health-
652 care buildings). Jan Sundell has fought for decades to put in place this OVK in Sweden, but he
653 mentions in his last editorial letter [67] that it is not enough. HVAC engineers must be properly
654 educated to the question of the IAQ, and its public health issues. He wrote in 2019 "*today in the*
655 *United States or China, students are not taught properly about ventilation. They are taught to design*
656 *air conditioning!!!*"

657 On the short term, either for natural or mechanical ventilations, increasing **their flow rate**
658 **should be achieved** when possible. This could be done by slight opening of windows if necessary. In
659 2009, Nielsen [68] analyzed experimentally the transport process of particles and tracer gases and
660 show that a high flow rate (i.e., an air change per hour ACH from 6 to 12 h⁻¹) to the ventilated space
661 reduces the level of viruses and bacteria in this space, without draught effect if sufficiently large
662 supply areas are used. The increased energy cost has to be put in balance with the considerable cost
663 (and economy impact) of present public policy in most countries. This is particularly true in public
664 buildings.

665 In some cases like offices, classrooms, aircraft or cabins, where people stand mostly at the
666 same desk/place, solutions as personalized, or piston ventilation [69], could be adopted.
667 Computational Fluid Dynamics have shown recently that personalized ventilation performed the best
668 to prevent cross-infection [70] compared to mixing ventilation, followed by displacement ventilation,
669 impinging jet ventilation, stratum ventilation and wall attachment ventilation.

670 As discussed previously, sterilizing and filtering air has the same effect than fresh air
671 ventilation. In his book of 1955 [4], Wells recommended a ventilation rate per pupil at school of 510
672 m³/h **which is an enormous value**, an order of magnitude higher than any current norm. Such flow
673 rates imply an important energy consumption. Probably aware of this difficulty, Wells proposed a
674 variety of solutions to sterilize air, and more particularly the use of UV lamps. Nowadays the
675 insufficient ventilation of schools and nurseries is largely recognized [71].

676 In order to remove infectious particles of air, HEPA (High Efficiency Particulate Air) filters could
677 be used. HEPA air filters can theoretically remove at least 99.97% of airborne particles with a size of
678 0.3 microns (µm). For efficient operation, the filters should be inspected quite regularly, and changed
679 periodically. A clogged HEPA filter can have a large leak rate through the peripheral gasket [72]. The
680 pressure drop through the filter can result in rather large energy consumption, beside the cost of
681 system equipment and maintenance.

682 The COVID-19 crisis has led to a considerable development of air purifiers and sterilizers that
683 use, amongst others, UV germicidal power, which is well documented for viruses [73]. It can be
684 shown by calculation that the UV power required to efficiently sterilize large air flow rates is rather
685 small [74]. Unfortunately, most of the sterilizer systems found on the market treat a much too low air
686 flow rate. The reason is probably that generally this kind of apparatus includes functions such as VOC
687 (Volatile Organic Compounds) treatment, and HEPA filters which results in higher costs.

688 Therefore, development of **cheap air sterilization units of very high flux** is clearly needed on
689 the mid-term. It is worth noting however, that employing such devices will make CO₂ diagnostics no
690 more relevant since the proportionality of the active virus concentration to the CO₂ one will not hold
691 anymore.

692 The way of life itself has implications on the disease transmission. More than a year ago Rowe
693 *et al.* made the prediction [75] that sub-Saharan Africa will not be stricken so much by the pandemic
694 in the future due to airborne considerations. This low spreading of the disease has been observed up
695 to now and a variety of explanations have been proposed [76,77]. Rowe *et al.* [6] have rationalized
696 this observation considering an "outdoor" way of life in these countries, which includes housing
697 without air conditioning (AC), with large natural ventilation to ensure refreshment and open outdoor
698 markets instead of supermarkets. South Africa where the prevalence of AC is much higher has been
699 more stricken and COVID-19 clusters have occurred there in closed supermarkets, most often
700 equipped with AC [78].

701 Therefore, it can be thought that in many places of low latitude, like West Indies or Guyana,
702 coming back as far as possible to the outdoor way of life could have immediate benefits. This
703 necessitates **waiving of AC when possible and turning back to natural cooling**, which implies large
704 current of fresh air. In many locations where heating cannot be avoided implying indoor way of life,
705 besides increasing ventilation, outdoor activities (for example outdoor markets) should be
706 encouraged.

707 The cheapest way to monitor pathogen IAQ is measurement of carbon dioxide concentration.
708 Too often, The concentration level alone is used as a sufficient risk proxy, and a limit around 800 ppm
709 has been proposed [79] as safe. We have shown throughout the present paper that communication
710 on this limit is misleading, as it ignores completely the question of the **time of exposure**. We propose
711 the development of **intelligent sensors** that could provide several integrated values of carbon
712 dioxide concentrations. Time of exposure and mean concentration, as defined by equation (21),
713 could be displayed by such sensors.

714 Last, close contact risk (except intimate i.e. < 0.6 m) is recognized as essentially airborne with
715 again a key role of exposure time [37]. In many situations, contact between two individuals lasts less
716 than fifteen minutes [80]. In this context, the risk drops to a very small value as soon as social
717 distancing between individuals is higher than 1.5 m, correspondingly to the communication of
718 government and health agencies. However, a misunderstanding of the real mode of transmission in
719 this case has led to irrational measures such as organizing files in supermarket with obligation to use
720 entrances different from exit. Although it has not been yet studied in the literature, staying in the
721 wake of an infector in a file for several minutes is certainly riskier than crossing the infector. We
722 recommend that, although social distancing must be encouraged, **such measures** directed against
723 fast crossing **should be removed** since they are misleading for the public and could in fact induce
724 higher airborne transmission.

725 5.2 Implications for interventions that are not directly targeting IAQ

726 The most radical intervention to mitigate the pandemic has certainly been the various forms of
727 lockdowns that, notably in western societies, constitute a major limitation to liberties and was
728 unprecedented in non-war conditions. While first lockdowns might have been necessary, given the
729 lack of governmental readiness to fight such pandemics in western societies, we now realize that,
730 beyond the obvious socio-economic implications, it has a significant downside related to
731 psychological isolation and mental health. Poorer families, children, women, and people experiencing
732 mental disorders have been particularly harmed by lockdowns [81-83], and this measure should be
733 taken in only the most extreme circumstances. Moreover, the efficiency of lockdown strategies is a
734 matter of debates [84,85]. Deleterious effects on people and families who live in small apartments
735 and closed places where IAQ is low is clear: **gathering people that have not been tested** in an indoor
736 housing **for a long time could be very counter-productive**: it has been shown indeed that a large
737 number of contaminations occur in family environment [86,87].

738 Far less radical, although very recent, contact-tracing apps on smartphones are the typical
739 intervention that any digital policy would have considered to support health policy. Such apps were
740 first introduced to help policy to fight the very lethal Ebola disease. However, their efficiency is
741 dubious and their ethical character questionable. When air monitoring measures, discussed in
742 previous sections, indicate a significant risk, the public should be informed in appropriate ways so
743 that behaviors can be adjusted. For risk induced by aerosol-based transmission, intuitive and
744 responsive user interfaces could be developed to visualize outbreak risks in various room of buildings
745 and alert facility managers and users in a way that could be similar and complement that outbreak

746 risks related to fomite-based transmission [88]. But mitigation measures such as contact tracing apps
747 will have little effect against long range transmission by aerosols. These apps have not been designed
748 to fight this transmission path of the pandemic and aerosol transmission was ignored at their
749 inception. When aerosols are emitted from delta variant, it is the exhaled microdroplets
750 concentrations in a given space that creates the major risk. Focusing on close crossing (less than one
751 meter for more than 15 minutes as we did in France with stopCOVID) in a public space can be
752 dangerous because people can feel safe (at least feel being well informed with their app), when in
753 fact what they should be warned (possibly by their smartphone, but even better by public screens or
754 specific systems) is about the situation over IAQ. Therefore, given the airborne danger of delta
755 variant, we consider that contact tracing apps are inappropriate for at least three reasons: First, to be
756 effective they require that a very large share of the population uses them for contact tracing which
757 has been considered unrealistic [89] and is still the case. In fact, whereas contact tracing apps have
758 been redesigned to be less intrusive (e.g Norway case) and their governmental communication to
759 influence their adoption adapted in to be less coercive (e.g. France case), a common nudging tactic to
760 influence their adoption has consisted in adding a number of features influencing individual benefits
761 such as giving information about risky regions or allowing to show conformity to vaccination plans to
762 access public places thus transforming a risk detection app into an information public health and a
763 sanitary pass app. As a result, after vaccination campaigns, these apps have been hugely
764 downloaded. However, the effective activation of the apps for personal risk detection is still very low.
765 Second, as we emphasize in the present paper, relevant parameters, **notably time of exposure to**
766 **risk**, and space, but not necessarily distance, to a likely infector, were not well understood by the
767 project developers [90,91]. Typically, distance for technology such as bluetooth is critical for accuracy
768 and reliability [90], but if the risk is related to the nearly homogeneous spread of virus in a given
769 space, the issue is about detecting the level of risk in this space and not necessarily identifying the
770 smartphone of the closest infector. Third, such apps may both develop bad habits in the population
771 and creates another danger for increasing potential discrimination and problems [89] such as fear of
772 mass surveillance as in Germany or Switzerland [92]. Conversely if major public spaces are equipped
773 with air monitoring equipment – currently monitoring CO₂ as proxy - that display public information
774 about IAQ, contact tracing apps would not be needed. Such information would be permanently
775 visible by the public on some fixed screens similar to clocks in such places. In addition, to increase
776 their situational awareness [93], those who are or feel potentially at risk could check the safety of
777 places on their smartphones by accessing a public website where measures of all displays would be
778 available on a map with color indicators. Both of these solutions would require people being
779 proactive. As the situation may vary a lot from place to place or evolve rapidly, the population at risk
780 could also use a warning emergency system conveying alerts through a dedicated device [94] or
781 some augmented reality app [88]. Given our experimentation measures, such public displays would
782 be highly trustworthy thanks to their high representational fidelity (notably current, nearly exact and
783 relevant (on these notions see [93])) of the CO₂ measures and thus limit the use of such warning
784 emergency systems to those at risk and not coerce all the population to acquiesce to a rampant form
785 of data surveillance. The cost of such air monitoring equipment and public website will not be very
786 high and they are a more ethical and scientifically valid choice, given the prevalence of the aerosol
787 transmission path, than current digital policy based on smartphone close-contact tracing.

788 **6 Conclusions**

789 The present health policies in many countries suffer from an original sin which was the deny of
790 airborne transmission. The advent of strains such as δ or \omicron leads to much higher quantum production
791 rates, implying that spreading of the epidemic is now mainly airborne. However, the communication
792 of most public authorities remains essentially directed toward avoiding close contact and fomites

793 transmission. Even if the importance of ventilation and mask wearing is now acknowledged, strong
794 decisions devoted to fight airborne transmission are not yet there. This is regrettable since some
795 mitigations measures in this field will not negatively impact people life, as others such as lockdown.

796 Major implications for public health policies have not been drawn from the conclusion that
797 new variants lead to dramatically airborne contamination. This is a significant conclusion of the
798 present paper on which we draw attention. Following the approach of Wells [4,5] and of most recent
799 researches ([36,37] amongst others) we derive simple formulas allowing to estimate risks in a variety
800 of situations. Applied to some specific observations of CO₂ level in a variety of environments they
801 highlight the importance of **the time of exposure** in risky situations.

802 Another major contribution of this paper is to highlight several interventions that need to be
803 introduced, modified, or could be suppressed. Some measures can be immediately taken at minor
804 costs, such as increasing ventilation when heating and using natural cooling in hot countries, coupled
805 to **CO₂ monitoring** to bring back CO₂ concentration to a satisfying level for the time of exposure.

806 We have shown that the ventilation systems, either natural or mechanical are often far of
807 following norms that are already insufficient. Therefore, **ventilation checking** should be promoted,
808 and **norms need to be revised** to include risk of pathogen transmission. Norms **must include**
809 **sterilizers** able to recirculate large air flows and which need to be developed at a reasonable cost.

810 In the short term, even if these measures are costly, a first plan to implement them in places
811 where public services are crucial such as hospitals and medical services [95,96], schools [97] is
812 necessary [98]. **Notably it must be clearly communicated that risk is not only dependent on CO₂**
813 **level, but also to the probability that an infector is or has been in the room and to the time of**
814 **exposure.**

815 Finally, digital means should be directed at informing people (e.g. with appropriate screens or
816 web applications possibly using augmented reality for particularly vulnerable persons, rather than
817 digitally tracing their (social) behavior and surveilling them). With the introduction of smartphone-
818 based contact-tracing apps further embedded in sanitary passes, the pandemic has considerably
819 accelerated the pace of the transformation of western societies towards digital surveillance. While
820 some initial intentions were hoped to be good, such trend is dangerous and shows that ethical use of
821 the digital is still in its infancy.

822 We insist that thinking only in terms of social distancing or social interactions has become a
823 paradigm that needs to be changed. Scientific literature demonstrates that we can be infected by
824 close contact, but other situations can be dangerous due to the very nature of airborne transmission.
825 As viruses can stay infectious in the air, we should not only consider the possibility of contamination
826 in co-presence, typically when people face each other, but also when people follow each other in a
827 file or even when infected people have left a poorly ventilated room. These scenarios need to be
828 highlighted in public information.

829 And last but not least, when the present pandemic will be over, what will stay in the mid and
830 long term is the necessity to **change our mind and norms in matter of IAQ**, in order to include this
831 problem of airborne pathogen transmission, an enormous challenge for building technology.

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838 Declaration of competing interest:

839 The authors declare that they have no known competing financial interests or personal relationships
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846 Author contributions

847 BR initiated, conceptualized and led the study. BR, AC and AM did the measurements described in
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849 reviewing of the paper.

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1145
1146

1147 Increased airborne transmission of COVID-19 with new variants.

1148 Implications for health policies.

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1150

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1153

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1165 **Supplementary materials**

1166

1167 SM1- Host entry characteristics:

1168 As discussed in the main paper the quantum of contagium, as defined by Wells [1], considers a
1169 variety of mechanisms including pathogen inhibition by host defenses. These defenses include,
1170 beside microbiological phenomena (immune response and others), some physical processes
1171 described below that are important for contamination by the aerosol route.

1172 In a series of remarkable experiments with rabbits and mice, Wells demonstrated that,
1173 concerning aerosols, very fine particles (which include dry nuclei) have a much higher infectious
1174 power than coarse particle, at least for disease such as tuberculosis and influenza. Wells' explanation
1175 was that the human body has a very efficient system to prevent coarse particle larger than a few
1176 micrometers to penetrate deep in the respiratory system. Beside defenses against very coarse
1177 particles, specific to the upper respiratory tract (nostrils, nasal cavity, mouth, throat, pharynx), and
1178 voice box (larynx)), mucociliary clearance is a primary innate defense mechanism of the lung (see the
1179 reviews by Bustamante-Marin and Ostrowski [2] and Kuek [3]) that helps to remove smaller particles
1180 and pathogens from the lower respiratory tract, using the epithelium formed by ciliated and
1181 secretory cells. These later provide a mucus which is expelled by cilia toward the digestive system
1182 after swallowing. It is known that most respirable pathogens do not provoke illness when ingested,
1183 and there is currently no evidence that COVID-19 could be transmitted by ingestion [4]. Note that the
1184 mechanism of very fine particles deposition into the lungs has been the subject of numerous studies
1185 for mineral toxic dusts, such as asbestos [5].

1186 Nowadays, the formidable progress of microbiology allows studying the influence of cellular
1187 characteristics on the vulnerability of cells to coronaviruses, which start with binding of the viral
1188 spike (S) proteins to cellular receptors [6]. Following some data, it has been anticipated that
1189 infectivity was higher in the upper respiratory tract and that the nose was a primary target [7].
1190 However the severity of the COVID-19 is linked to the occurrence of pneumonia, followed by acute
1191 diffuse alveolar damage, which can be due to direct lung infection by airborne microparticles [8,9] or
1192 by indirect infection from the oropharynx to the lung by aspiration of the viral inoculum when
1193 breathing [7]. Also the study of nonhuman primate model reveals, after autopsy, the importance of
1194 lung lesions in macaques [10]. It seems reasonable to assume that, when the virus reaches the lungs
1195 directly, before some immunity able to inhibit viral reproduction has been acquired, it could result in
1196 devastating pneumonia, as sometimes reported in young, healthy subjects.

1197 It has to be noticed that as well the remarkable experimental results of Wells for particle size
1198 than the most recent findings of microbiology cannot be directly used to develop a quantitative
1199 model of transmission risk. Therefore, some concepts and approaches must be developed prior to
1200 the establishment of any risk model.

1201

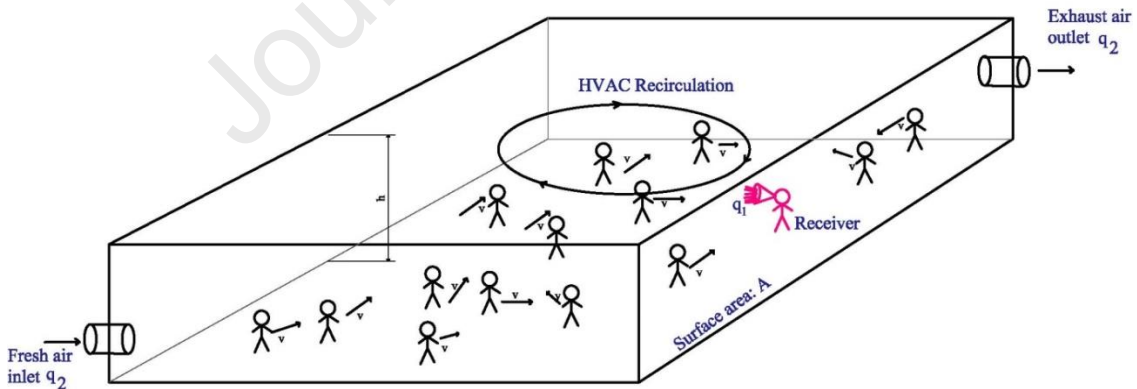
1202 **SM2- Conservation and transport equations**

1203 It is far beyond the possibility of this section of the supplementary materials to develop the
 1204 complexity of transport and conservation equations for diphasic turbulent fluids, with the target of
 1205 precise calculations of the fields of velocities, temperature and concentrations of the various
 1206 components. We shall just present the equation used in the main paper for the case of a well-mixed
 1207 room (homogeneous hypothesis) and the approach underlying much of the calculations used in
 1208 inhomogeneous models in order to calculate the concentration field of infectious particles.

1209 In a homogeneous model, it is assumed that there is no spatial gradient of risk in a space
 1210 where the infectors and the receivers either evolve or stay in place. In other words, it is assumed that
 1211 the infectious microdroplets are evenly distributed. This is typical of two kinds of situations. It
 1212 happens first instantly in a space where high performance mixing ventilation is achieved using special
 1213 air terminal units designed to promote a high jet induction (i.e., vortex diffusers, lobed diffusers).
 1214 This case lies to forced convection state. In absence of this kind of mixing ventilation there are a
 1215 variety of air motions induced by other phenomena, such as natural convection, wake of moving
 1216 people, door openings for letting people in or out. It can be shown that in many situations of this
 1217 sort, the well mixed room hypothesis is also valid [11]. Then, we consider an evenly distribution of
 1218 microdroplets obtained by induced turbulent flows, although this distribution is not really continuous
 1219 due to its discrete character (very low concentration). Using CO₂ as a proxy of infectious
 1220 microdroplets (i.e. quanta), observations show that this condition is most often fulfilled (see main
 1221 paper).

1222 Of course, if specific ventilation techniques are used [12], the generated directional air flows
 1223 within the room lead to preferential aerosols trajectories following air distribution patterns.

1224 In a homogeneous model it is possible to write a conservation equation for the concentration
 1225 n_i of mono-sized microdroplets in a volume V , as developed by Rowe et al [13] for the indoor risk
 1226 assessment, to compare with the outdoor case. Figure SM2-1 depicts the situation:



1227
 1228 Figure SM2-1: a typical indoor homogeneous situation.

1229 In this figure the inlet and outlet ventilation flow rates are assumed equal with the value q_2 .
 1230 Let N_p be the number of people inside, $N_i(t)$ the total number of aerosol particles of human
 1231 respiratory origin inside the volume, resulting in a concentration of particles of $n_i(t) = N_i(t)/V$. The
 1232 mean exhaled flow rate of a person was taken as p (of course identical to the inhaled rate) and the
 1233 concentration of particles in this flow was assumed equal to n_1

1234 (SM2-1)
$$\frac{dN_i}{dt} = V \times \frac{dn_i}{dt} = N_p \times p \times n_1 - q_2 \times n_i$$

1235 It was assumed no sink term for the particles inside the volume.

1236 In the same way, an equation of conservation can be applied to the quanta of contagium as
 1237 defined by Wells [1]. Let N_q be the total number of quanta in the volume V and n_q the quantum
 1238 concentration. Considering the quantum production rate per infector q and introducing a quantum
 1239 lifetime, which can be considered as the virus lifetime, τ_i , this equation reads:

$$1240 \quad (\text{SM2-2}) \quad \frac{dN_q}{dt} = V \times \frac{dn_q}{dt} = I \times q - q_2 \times n_q - \frac{n_q}{\tau_i} \times V$$

1241 In this equation we consider the number of infectors I within the volume since only infectors emit
 1242 quanta.

1243 Assuming $n_q(0) = 0$, The solution of (SM2-2) is:

$$1244 \quad (\text{SM2-3}) \quad n_q(t) = n_q^\infty \times \left[1 - \exp\left(-\frac{t}{\tau_1}\right) \right]$$

1245 with:

$$1246 \quad (\text{SM2-4}) \quad \tau_1 = \frac{V}{q_2 + \frac{V}{\tau_i}}$$

1247 The concentration of quanta at stationary state i.e. $t \sim a \text{ few } \tau_1$ is:

1248

$$1249 \quad (\text{SM2-5}) \quad n_q^\infty = \frac{I \times q}{\left(q_2 + \frac{V}{\tau_i}\right)}$$

1250 which, if the virus lifetime is neglected, reduces to:

$$1251 \quad (\text{SM2-6}) \quad n_q^\infty = \frac{I \times q}{q_2}$$

1252 Note that if a device able to sterilize a flow rate q_3 is used, the above equations hold just by
 1253 replacing q_2 by $Q = q_2 + q_3$.

1254 These equations founded on the well mixed room hypothesis are the basis of the famous Wells-
 1255 Riley model and are convenient for a very large number of indoor situations. However,
 1256 inhomogeneous infection patterns are reported for a number of well-documented transmission
 1257 events in closed spaces, especially in restaurants [14-16] but also in other places such as aircrafts
 1258 [17]. Generally, in these specific well studied cases, inhomogeneity was created by the mechanical
 1259 ventilation system of air conditioning (hereafter AC) with recirculation, inducing locally larger air
 1260 velocity. One typical and largely mediatized event concerned a restaurant in Guangzhou, China. It has
 1261 been the subject of numerical modeling [14]. Numerous published works in the field do not relate to
 1262 a specific observed event but to hypothetical situations supposed to represent typical cases, such as
 1263 a supermarket [18]. These models rely on CFD (Computational Fluid Dynamics) calculations of the air
 1264 flow stream, using a variety of software, such as Open Foam for example. Then the microparticle
 1265 behavior is estimated using a variety of methods (Lagrangian, Monte-Carlo). In the Lagrangian
 1266 approach the movement of each particle is calculated using Newton's second law of motion, where,
 1267 within forces acting on the particle, the drag one is determined from the calculated field of air
 1268 velocity. Note that, for a Stokes number $\ll 1$, the particles are just assumed to follow the flow. The
 1269 Stokes number can be defined as the ratio of two times τ_a/τ_h , τ_a being the time of velocity
 1270 accommodation of a particle to the flow velocity and τ_h the hydrodynamic time (equal to a typical
 1271 length of the problem divided by the flow velocity). The Stokes number reads [19]:

1272

1273

$$St = \frac{D_p^2 \times \rho_p \times C_c \times u_0}{18 \times \mu \times L}$$

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With D_p and ρ_p being respectively the diameter and volume mass of the particle, μ the air viscosity, u_0 and L respectively a typical order of magnitude of flow velocity and length. C_c is a slip parameter which takes into account the value of the particle Knudsen number. For particles of the size considered in airborne transmission C_c is very close to one. Note that L/u_0 is the hydrodynamic time and that for most problems dealing with the behavior of exhaled aerosol particles in indoor situation the Stokes number remains much smaller than one, except for large particles in the close contact case discussed in next section.

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It is also worthwhile to point out that when inhomogeneous infection modeling is applied to a specific geometry of the environment, it can be applied as such for the design of a new building for example but is limited for applications in the real life of most existing buildings and therefore, on the short term, for driving public policy. What is more interesting is the modeling of airborne close contact discussed in the next section.

1286

1287 **SM3- Close contact transmissions**

1288 It is now largely admitted that the transmission of COVID-19 disease by close contact is most
1289 often an airborne one, referred in the literature as “short-range airborne transmission”
1290 [20,20,21,21]. Close to the emitter the turbulent expiratory plume (or puff for cough and sneeze) can
1291 have a much higher quantum (viral) load than in the ambient air of the indoor space considered.
1292 Several models of this phenomena have been proposed, some very simple [20] others more
1293 sophisticated. The recent one by Cortellessa *et al.* [21] employs CFD for the air flow and Lagrangian
1294 calculations for the particles to derive the dose and the risk as a function of the distance between
1295 infector and susceptible. Not only the distance but also the time of exposure is considered in order to
1296 assess the risk, although the time is limited to fifteen minutes. Large microdroplets which behave in a
1297 ballistic way are also considered and shown to prevail only at very short distance (< 60 cm), with a
1298 contribution to the dose being completely negligible at higher distances, demonstrating the airborne
1299 character of most airborne contamination in close contact, excepted intimate.

1300 In their paper, Cortellessa *et al.* also made a comparison with the homogeneous risk. However,
1301 the comparison is restricted to the same time of exposure of fifteen minutes, with an initial
1302 concentration of quanta equal to zero. Therefore, it does not consider long times of exposure for the
1303 homogeneous case at steady state, as found for example in schools but such an extension can easily
1304 be done. Indeed, a good comparison should have to include the probability of close contacts
1305 together with contact durations, which is not done. Such a close contact risk assessment is anyway
1306 extremely useful for public policy.

1307

1308

1309

1310 **SM4- Infector proportion and combination analysis**

1311 The problem of determining the exact proportion r of infectors I in a large population N_{Tot}
 1312 ($r = I/N_{Tot}$) is a difficult one. Two statistical results are most often available. The positivity rate is
 1313 the number of populations tested positive related to the total number of people tested, and
 1314 therefore is a proportion without dimension. The incidence rate is the number of new people tested
 1315 positive in a population, which can then be reported to a target population (for example 10^5
 1316 individuals) for a given period of time (for example one day or one week). It is therefore a temporal
 1317 rate and, as such, has the dimension of (time)⁻¹. It is clear from these definitions that the results will
 1318 depend on which people are tested and also of the size of the target. Since many people are infected
 1319 but not tested and that people tested positive in the past remain infectious for some time, it can be
 1320 anticipated that the real number of infectors could be much higher than what can be deduced from
 1321 an analysis of the incidence rate: in principle, this rate can drop to zero with still infectors in the
 1322 population. Further, since the population tested is often a symptomatic one, the positivity rate of
 1323 testing could be much higher than the real proportion of infectors. Only a blind testing of a
 1324 representative population would lead a true value of r .

1325 Therefore the purpose of the present SM is just to show that it is possible to estimate the
 1326 probability of infection of a susceptible target using a simplified expression (see SM4-3) which
 1327 essentially considers the given proportion of infectors r in a population of N_{Tot} individuals, provided
 1328 that the ventilation flow rate per person, q_{norm} , is known and the time of exposure t is fixed. Here N_{Tot}
 1329 will represent the inhabitants of a country, a region, a metropole or a city or it can also denote a
 1330 fixed reference population like 100000, for instance. Then, N_{Tot} is large. The number of infected
 1331 people in that population will be quoted I further in the text (see SM4-6 and beyond) with $I = r \times N_{Tot}$.

1332 In the main paper we have derived an equation for the dose inhaled by a susceptible person:

1333 (SM4-1)
$$X = \frac{r \times p}{q_{norm}} \times q \times t$$

1334 which assumes that the total ventilation rate q_2 is given by $q_{norm} \times N_p$, where N_p is the number of
 1335 people present, with the susceptible target, in a specific location. Here $N_p \ll N_{Tot}$. It is also assumed
 1336 that the proportion of infected people r is also representative of the sanitary situation in the space of
 1337 interest. In other words, if n is the number of infectors in the restricted population of N_p persons, we
 1338 assume that $r = n/N_p = I/N_{Tot}$. We also remember that p and q are the respiratory flow rate and
 1339 the quantum rate of pathogens per infector expressed in h⁻¹, respectively.

1340 From this, the probability of infection is given by the Wells-Riley expression already presented in the
 1341 main text (eq. 3):

1342 (SM4-2)
$$P_{WR} = 1 - \exp(-X)$$

1343 or

1344 (SM4-3)
$$P_{WR} = 1 - \exp\left(-\frac{r p}{q_{norm}} q t\right)$$

1345 Another way to calculate this probability, which seems to be more realistic, is to make a weighted
 1346 summation of probabilities to be infected in conditions where one, two, three etc. infectors are
 1347 present in the restricted population of N_p people. This can be expressed as:

1348 (SM4-4)
$$P = \sum_1^{N_p} P_n(r) \times P_{WR}(n)$$

1349 where $P_n(r)$ is the probability to have n infectors and $P_{WR}(n)$ the Wells Riley probability of being
 1350 infected with n infectors in the population of N_p individuals. Then:

1351 (SM4-5)
$$P_{WR}(n) = 1 - \exp\left(-\frac{np}{q_2}qt\right)$$

1352 This new expression SM4-4 has an interesting advantage with respect to the simpler equation SM4-3
 1353 since it discriminates the individual $P_n(r)$ contributions from each other. Then, it is possible to
 1354 evaluate how significant is each term in the summation and more particularly if the state with only
 1355 one infector can be representative of the total risk of infection or not.

1356 Probability $P_n(r)$ is dependent on the number of infected people I and consequently it is also a
 1357 function of r . It can be derived from a combinatory analysis. Defining $C_{N_{Tot}}^{N_p}$ as the number of
 1358 combinations of selecting an ensemble of N_p persons in a larger group of N_{Tot} individuals, one can
 1359 express the number of combinations that include n individuals with a given property (here infection)
 1360 in the selected group of N_p people. Then the probability of having n individuals infected in the
 1361 restricted population N_p is simply given by:

1362 (SM4-6)
$$P_n(r) = \frac{C_I^n C_{N_{Tot}-I}^{N_p-n}}{C_{N_{Tot}}^{N_p}}$$

1363 We remember here that the number of combinations of i elements in a global ensemble of j objects
 1364 (with $j \geq i$) is mathematically equal to:

1365 (SM4-7)
$$C_j^i = \frac{(j)!}{(i)!(j-i)!}$$

1366 From this, equation SM4-4 becomes:

1367 (SM4-8)
$$P = \sum_1^{N_p} \frac{C_I^n C_{N_{Tot}-I}^{N_p-n}}{C_{N_{Tot}}^{N_p}} \times \left(1 - \exp\left(-\frac{np}{q_2}qt\right)\right)$$

1368 This expression is numerically evaluated below for a few examples and compared to equation SM4-3.
 1369 We consider here situations for which the restricted population is smaller than the total number of
 1370 infectors in the reference population N_{Tot} :

1371 (SM4-9)
$$N_p < I$$

1372 Calculations are be made considering a reference population N_{Tot} of 10^5 ; a respiratory flow rate p of
 1373 $0.5 \text{ m}^3/\text{h}$; a quantum infection rate q of 40 h^{-1} and a time of exposure t of 2 hours. A standard
 1374 ventilation flow rate q_{norm} of $20 \text{ m}^3/\text{h}/\text{person}$ will be also employed. The proportion of infected
 1375 people r is varied between 0.001 and 0.03 and the restricted population N_p is chosen as either 80 or
 1376 30. From this, the number of infected people in the N_{Tot} main group will vary from 100 to 3000
 1377 according to the r value, thus respecting inequality SM4-9.

1378 Results of SM4-3 and SM4-8 are presented in Table SM4-1 and SM4-2 for the two values of N_p . In
 1379 addition, we indicate the limit of n , quoted n_{cut} , beyond which $P_n(r) \times P_{WR}(n)$ terms do not
 1380 contribute significantly to the summation in SM4-8; the value of n , quoted n_{max} , corresponding to the
 1381 main contribution $P_{n_{max}}(r) \times P_{WR}(n_{max})$ in the summation and the percentage of this contribution
 1382 to P value.

1383

1384

1385 Table SM4-1: Comparison of P_{WR} with P for a restricted population N_p of 80 individuals

r	P_{WR} (SM4-3) %	P (SM4-8) %	n_{cut}	n_{max}	n_{max} contribution %
0.001	0.200	0.197	2	1	93
0.003	0.598	0.591	3	1	79
0.010	1.980	1.956	4	1	46
0.030	5.824	5.756	8	3	27

1386

1387 Table SM4-2: Comparison of P_{WR} with P for a restricted population N_p of 30 individuals

r	P_{WR} (SM4-3) %	P (SM4-8) %	n_{cut}	n_{max}	n_{max} contribution %
0.001	0.200	0.193	2	1	97
0.003	0.598	0.579	2	1	92
0.010	1.980	1.917	3	1	75
0.030	5.824	5.644	5	1	43

1388

1389 These calculations demonstrate a very good agreement between both ways of determining the
1390 probability of infection from either P_{WR} or P . The agreement is even better when the restricted
1391 population is enhanced, essentially due to the statistical effect of using larger N_p numbers. It can also
1392 be shown that the contribution of one infector ($n_{max}=1$) in the summation is the main one in many
1393 situations although, however, summation cannot be limited to the first term in SM4-8 for several
1394 conditions as indicated by the n_{cut} value and the " n_{max} contribution" columns. The lower the
1395 proportion of infectors r , the larger the contribution of $P_1(r) \times P_{WR}(1)$ which makes a lot of sense
1396 since for small r the probability of having more than one infector in the restricted population N_p
1397 becomes very small.

1398 To conclude we stress that we have restricted the demonstration to a limited number of
1399 configurations but it is worth pointing out that several parameters act in a similar way
1400 mathematically speaking. Then, changing the time of exposure or/and the quantum rate of infectors
1401 would lead to essentially the same kind of conclusions.

1402

1403 **SM5- Masks, quantum production rate and inhaled dose**

1404 To build a probabilistic model of infection it is necessary to know the production rate of quanta
 1405 (as defined by Wells) by an infector. It is defined per unit time and per infector (unit: h^{-1} for example)
 1406 and can be deduced from epidemiological observations [22] but also linked to the distributions of
 1407 microdroplets emitted by humans, together with the knowledge of viral load in respiratory fluids and
 1408 of the mean number of viruses required to infect 63% of susceptibles.

1409 As stated in the main paper and following Buonanno *et al.* [23], the quantum production rate q
 1410 can be written as:

$$1411 \text{ (SM5-1)} \quad q = VL \times c \times p \times \int_0^{10\mu m} N_d(D) \times dV_d(D)$$

1412 where VL is the viral load in the respiratory fluid, c is a factor of proportionality between the viral
 1413 content (copies/unit volume) and quanta, p is the pulmonary exhaled volume rate (volume/unit
 1414 time), $N_d(D)$ the size distribution of droplets (diameter D) of volume V_d .

1415 Morawska *et al.* [24] have shown that microdroplets emitted by different expiratory activity
 1416 correspond to four different modes of size distribution, centered on mid-point diameters of
 1417 respectively $D_1 = 0.8$, $D_2 = 1.8$, $D_3 = 3.5$, and $D_4 = 5.5 \mu m$. Their concentrations depend on the
 1418 expiratory activity as shown in table SM5-1 adapted from Table 1 of Buonanno *et al.* [23]:

1419 Table SM5-1: Concentrations (in cm^{-3}) of the microdroplets size modes during various expiratory
 1420 activities

Expiratory activity	Centered mid-point diameter (μm)			
	0.80	1.8	3.5	5.5
Voiced counting	0.236	0.068	0.007	0.011
Whispered counting	0.110	0.014	0.004	0.002
Unmodulated vocalization	0.751	0.139	0.139	0.059
Breathing	0.084	0.009	0.003	0.002

1421

1422 It results that equation (SM5-1) can be simplified as:

$$1423 \text{ (SM5-2)} \quad q_j = VL \times c \times p \times \sum_{i=1}^{i=4} N_{ij} \times V_i$$

1424 where the subscripts i and j refer to the size mode and the expiratory activity respectively.

1425 From equation SM5-2 and Table SM5-1 it is clear that the production rate of quanta can vary
 1426 widely depending on the expiratory activity but also on the virus strain through VL and c . Note also
 1427 that the level of activity (which implies a given metabolism) plays an important role on this rate [23].
 1428 Therefore, it can change with time for a given infector.

1429 For a given respiratory activity, equation (SM5-2) can be written as:

$$1430 \text{ (SM5-3)} \quad q = \sum_{i=1}^{i=4} q_i$$

1431 where the subscript j has been omitted.

1432 In the absence of masks for the emitter (infector) and the receiver (susceptible) the dose
 1433 inhaled by the receiver can be written:

$$1434 \text{ (SM5-4)} \quad X = \int_0^t n_q^\infty \times p \times dt$$

1435 where n_q^∞ is given by equation SM2-6.

1436 When a mask is worn the proportion of particles going through the mask could be strongly
 1437 dependent of the particle size. Therefore, it could be considered that the quantum production rate is
 1438 reduced accordingly and that it is possible to define a quantum production rate depending on the
 1439 mode:

1440 (SM5-5)
$$q_i' = \beta_i \times q_i$$

1441 As a conservation equation can be written for each mode, a concentration of quantum for this
 1442 mode at stationary state will result:

1443 (SM5-6)
$$n_{q,i}^\infty = \frac{I \times \beta_i \times q_i}{q_2}$$

1444 If the receiver wears the same kind of masks the inhaled dose of this mode of particles should be:

1445 (SM5-7)
$$X_i = \beta_i \times \int_0^t n_{q,i}^\infty \times p \times dt = \beta_i^2 \times \int_0^t \frac{I \times q_i}{q_2} \times p \times dt$$

1446 Then the total dose would be:

1447 (SM5-8)
$$X = \sum_i X_i$$

1448 For the smallest size, below $1 \mu\text{m}$ ($i = 1$), the surgical mask can be very inefficient as shown by
 1449 [25,26] leading to a value of β_i close to 0.5 for the flow through the filtration media.

1450 However due to the importance of the leaks [27], it could be assumed that β_i is also very large
 1451 even for particles larger than $1 \mu\text{m}$ (except for the largest ones which behave in a ballistic way and
 1452 are completely trapped). Then, using equations SM5-7 and SM5-8 with the results of [26,27], it can
 1453 be shown that wearing the mask reduces the quantum production rate by a factor of three. As the
 1454 dose of inhaled particles is reduced by the same factor, an overall efficiency in dose reduction of
 1455 around 90% can be assumed if emitters and receivers wear it, as it has been assumed for schools in
 1456 the main paper.

1457

1458

1459

1460 **SM6- The ICONE index**

1461 Based on indoor CO₂ concentrations, the ICONE air stuffiness index [28] has been developed in 2008
 1462 by the French Scientific and Technical Center of Building (CSTB) especially for IAQ evaluation in
 1463 schools. In 2012, the ICONE air stuffiness index has been integrated into the framework for the
 1464 mandatory monitoring of IAQ in some public buildings in France (IAQ decree n° 2012-14 [29]). The
 1465 ICONE index takes into account the frequency and intensity of CO₂ levels around the threshold
 1466 values of 1000 and 1700 ppm during normal occupancy of the classroom by children. The
 1467 confinement level is then expressed by a score scaled in six levels from 0 to 5. The score 0
 1468 corresponds to zero confinement (CO₂ level always below 1000 ppm), this is the most favourable
 1469 situation. Notes 2 and 3 correspond to low and regular confinement, whereas notes 4 and 5
 1470 correspond to very high and extreme confinement, level 5 is the most unfavourable situation (CO₂
 1471 concentration always above 1700 ppm during occupancy). In this case, the decree [29] stipulates that
 1472 additional investigations must be carried out and the local authority (the departmental Prefect) must
 1473 be informed. Table below summarizes the various situations:

ICONE	Stuffiness level
0	None
1	Weak
2	Moderate
3	High
4	Very high
5	extreme

1474

1475 The icone index can be calculated precisely using the following expression:

$$1476 \quad \text{ICONE} = 8.3 \log_{10}(1 + f_1 + 3 f_2)$$

1477 where f_1 and f_2 represent the proportions of CO₂ concentration measurements comprised in between
 1478 1000 and 1700 ppm or higher than 1700 ppm respectively. Hence, the ICONE index is zero when all
 1479 measurements have been found below 1000 ppm ($f_1 = f_2 = 0$) as said earlier whereas it is 5 when all
 1480 measurements are higher than 1700 ppm ($f_1 = 0$ and $f_2 = 1$).

1481

1482 **SM7- The concentration of carbon dioxide as a proxy of the quantum concentration**

1483 The exhaled breathing of human beings contains a much higher concentration of carbon
 1484 dioxide than the normal outdoor air. As a matter of consequence when persons are gathered in a
 1485 room this leads to a noticeable increase of its concentration as it was recognized by previous authors
 1486 [30]. Considering the situation depicted in figure SM2-1, a conservation equation for CO₂ can be
 1487 written in the same way than for particles or quanta:

$$1488 \quad (SM7-1) \quad V \times \frac{dCO_2}{dt} = N_p \times p \times CO_{2,exh} - q_2 \times \{CO_2 - CO_{2,ext}\}$$

1489 with the same notation meaning than in SM2 for V , N_p , p and q_2 . CO_2 is the current concentration of
 1490 CO₂ which can be expressed in ppm (part per million) since air density is assumed constant. $CO_{2,exh}$
 1491 and $CO_{2,ext}$ are respectively CO₂ concentration in the air exhaled by a human (close to 40 000 ppm)
 1492 and outdoor fresh air (around 420 ppm).

1493 The last term of the equation comes from the fact that the fresh outdoor air contains CO₂.

1494 It follows that the carbon dioxide concentration in the room, equal to $CO_2(0)$ at $t = 0$, will evolve
 1495 following the equation:

$$1496 \quad (SM7-2) \quad CO_2(t) - CO_2(0) = \frac{N_p \times p \times CO_{2,exh}}{q_2} \times \left[1 - \exp\left(-\frac{t}{\tau_2}\right) \right]$$

1497 with

$$1498 \quad (SM7-3) \quad \tau_2 = V/q_2$$

1499 Note that most often a “clean” room with a null virus concentration $n_q(0) = 0$ corresponds to
 1500 $CO_2(0) = CO_{2,ext}$, excepted in un-stationary conditions, for example if ventilation is off during the
 1501 night and considering a virus lifetime, see end of this SM.

1502 When the quantum (virus) lifetime is very large, τ_1 defined by equation SM2-4 reduces to τ_2 .
 1503 Then, at any time t , it is straightforward to deduce from equations SM2-3, SM2-5 and SM7-2 that:

$$1504 \quad (SM7-4) \quad \frac{n_q(t)}{CO_2(t) - CO_{2,ext}} = \frac{I \times q}{N_p \times p \times CO_{2,exh}}$$

1505 which assumes that at $t = 0$, $CO_2(0) = CO_{2,ext}$ and $n_q(0) = 0$.

1506 Note that it can be shown that the same equation holds for the poorly ventilated case developed in
 1507 the main paper.

1508 Then using the fact that the dose is:

$$1509 \quad (SM7-5) \quad X = \int_{t_0}^{t_1} n_q \times p \times dt$$

1510 It follows, for a time of exposure $\Delta t = t_1 - t_0$, that:

$$1511 \quad (SM7-6) \quad X = \frac{CO_{2,mean}}{CO_{2,exh}} \times r \times q \times \Delta t$$

1512 with:

$$1513 \quad (SM7-7) \quad CO_{2,mean} = \frac{\int_{t_0}^{t_1} \Delta CO_2(t) \times dt}{\Delta t} \quad \text{and} \quad \Delta CO_2(t) = CO_2(t) - CO_{2,ext}$$

1514 This equation was first established by Rudnick and Milton [30] in a different way and is valid
 1515 even for unstationary conditions as long as the virus lifetime $\tau_i \gg V/q_2$.

1516 If the above conditions for τ_i is not fulfilled it is necessary to write a new equation for the dose
 1517 as a function of time. Still assuming that at $t = 0$, $CO_2(0) = CO_{2,ext}$ and $n_q(0) = 0$, Equation SM7-
 1518 4 is changed as:

1519 (SM7-8)
$$\frac{n_q(t)}{CO_2(t) - CO_{2,ext}} = \frac{I \times q}{N_p \times p \times CO_{2,exh}} \times \frac{q_2}{(q_2 + V/\tau_i)} \times C(t)$$

1520 with:

1521 (SM7-9)
$$C(t) = \frac{\{1 - \exp(-t/\tau_1)\}}{\{1 - \exp(-t/\tau_2)\}}$$

1522 which at stationary state reduces to:

1523 (SM7-10)
$$\frac{n_q(t)}{CO_2(t) - CO_{2,ext}} = \frac{I \times q}{N_p \times p \times CO_{2,exh}} \times \frac{q_2}{(q_2 + V/\tau_i)}$$

1524 and for the dose at stationary state:

1525 (SM7-11)
$$X = \frac{CO_{2,mean}}{CO_{2,exh}} \times r \times q \times \Delta t \times \frac{q_2}{(q_2 + V/\tau_i)}$$

1526 Of course, for transient evolution the dose can be calculated using equation SM7-5 with SM7-8. Now
 1527 other transient evolutions could be considered with different initial conditions than the choice made
 1528 above. For example if the ventilation is off overnight the virus lifetime could be such that in the
 1529 morning ($t = 0$), the conditions $n_q(0) = 0$ holds but with $CO_2(0) > CO_{2,ext}$. In this case $CO_{2,ext}$
 1530 should be replaced by $CO_2(0)$ in equations SM7-8 and SM-7-10. These considerations show the
 1531 importance of the virus lifetime, which is strongly dependent of the room conditions, especially the
 1532 temperature [31]. Nevertheless, it remains that CO_2 is most often an excellent proxy of the risk,
 1533 excepted when an air sterilizer at high volume flow rate $q_3 \geq q_2$ is used.

1534

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Highlights

- *Relative airborne risk assessment following variant viral load and contagiousness*
- *Indoor analytical risk assessment including absence of ventilation*
- *Adequacy of the present norms of ventilation to Covid-19 pandemic*
- *Observation of non-compliance to standards concerning CO₂ Indoor Air Quality*

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Declaration of interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests:

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