Correlation of Respiratory Aerosols with Metabolic Carbon Dioxide

Niklas Kappelt1,2,*, Hugo S. Russell2,3,4, Szymon Kwiatkowski2, Alireza Afshari5, and Matthew S. Johnson1,2

1Department of Chemistry, University of Copenhagen, DK-2100 Copenhagen Ø, Denmark
2Airlabs, Nannasgade 28, DK-2200 Copenhagen N, Denmark
3Danish Big Data Centre for Environment and Health (BERTHA), Aarhus University, DK-4000 Roskilde, Denmark
4Department of Environmental Science, Aarhus University, DK-4000 Roskilde, Denmark
5Department of the Built Environment, Division of Sustainability, Energy and Indoor Environment, Aalborg University, DK-2450 Copenhagen SV, Denmark
*niklas.kappelt@gmail.com

ABSTRACT

Respiratory Aerosols from breathing and talking have found wide acceptance as a transmission route for viruses such as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Previous studies have found particles with diameters ranging from 10 nm to 145 µm, produced from different regions in the respiratory system. We present the first chamber study, in which respiratory aerosols have been simultaneously measured with carbon dioxide (CO₂) to establish the correlation between the two concentrations. CO₂ concentrations are easily available through low-cost sensors and could be used to estimate viral exposure through this correlation, whereas source-specific aerosol measurements are complicated and not possible with low cost sensors. The increase in both PM10 and CO₂ was linear over ten minutes in a 2 m³ chamber for all participants, suggesting a strong correlation. On average, talking released more particles than breathing, with 14,600 ± 16,800 min⁻¹ (one-σ standard deviation) and 6,210 ± 5,630 min⁻¹ on average, respectively, while CO₂ increased with 139 ± 33 ppm min⁻¹ during talking and 143 ± 29 ppm min⁻¹ during breathing. Assuming a typical viral load of 7 × 10⁶ RNA copies per ml of oral fluid, ten minutes of talking and breathing are estimated to produce 7 and 16 suspended RNA copies, respectively, correlating to a CO₂ concentration of around 1,800 ppm in a 2 m³ chamber. This provides a strong argument for keeping indoor spaces well ventilated and shows how CO₂ concentrations, measured with low-cost sensors, could be used as a proxy for viral exposure.

Introduction

A key reason for the difficulty of controlling the COVID-19 pandemic is the airborne spread of the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Particles are emitted from the human respiratory system not only by coughing and sneezing, but also by talking and breathing. These particles can remain airborne for hours and travel far outside the personal space of the emitter. SARS-CoV-2 has been found in these particles on several occasions and the virus remains viable in aerosol droplets for hours, with a half life of 1.1 h. Before the COVID-19 pandemic, similar airborne transmission properties were also found for SARS, MERS and influenza. Particle concentrations are typically expressed as either number of particles per air volume (m⁻³) or mass per air volume (µg m⁻³). With a known, average concentration of 7 × 10⁶ RNA copies ml⁻¹ of oral fluid, the volume concentration in ml m⁻³ is particularly of interest.

Generally, anthropogenically emitted pollutants are known as bioeffluents. Besides respiratory aerosols, these include carbon dioxide (CO₂), water vapour and volatile odorous substances. As CO₂ is the main pollutant and it is easy and cheap to measure, its concentration can be a good indicator of other anthropogenic pollutants in indoor spaces, as the only other sources would be open flames, pets or combustion devices. Because CO₂ concentrations increase with the number of occupants in a space, and one or more of the occupants could potentially be infected, it is logical that respiratory disease risk scales upwards with indoor CO₂ levels. Du et al., for example, demonstrated that when CO₂ was reduced to less than 1000 ppm, it was independently associated with a 97 % decrease in tuberculosis incidence among contacts.

In 2003, Rudnick and Milton introduced a model that uses CO₂ levels to estimate the risk of airborne transmission of influenza and other respiratory diseases. Peng and Jimenez used this model to estimate the COVID-19 infection risks for different scenarios and found that even though the CO₂ level corresponding to a given infection risk may vary by over two orders of magnitude depending on the environment and activity, it is still a useful metric to assess ventilation. During the COVID-19 pandemic, several researchers estimated the transmission risk of SARS-CoV-2 in such spaces as nail salons and office buildings based on occupant respiration rates and activities. For instance, Harrichandra et al. concluded that increased
outdoor airflow rates and the use of face masks by both employees and customers could substantially reduce SARS-CoV-2 transmission in New York City nail salons. These studies demonstrated the value of CO$_2$ sensing for assessing airflow as a factor in transmission risk.

Numerous studies directly investigated respiratory aerosols from breathing, talking and coughing and found particles in the range of 10 nm to 145 µm$^3$. We present the first chamber study investigating respiratory aerosols while simultaneously measuring metabolically produced carbon dioxide. The aim of our study was to find the correlation between the two pollutants and estimate the suspended fluid volume as well as the suspended SARS-CoV-2 RNA number of a hypothetical infected person at a given CO$_2$ concentration.

**Results**

**PM10 and CO$_2$ Increase over Time**

In a 2 m$^3$ chamber, particle and CO$_2$ concentrations were simultaneously measured over ten minutes while a subject was either breathing or talking. 16 volunteers (8 male, 8 female) participated, but one data set was excluded as corrupt due to an error. A comparison of the typical increase in PM10 (particulate matter with an aerodynamic diameter smaller than 10 µm) from background leakage and the breathing and talking experiments of one of the participants are shown in Figure 1A. The corresponding CO$_2$ concentrations over time are shown in Figure 1B.

![Figure 1](image-url)

**Figure 1.** Typical experimental data for one of the 15 participants. (A) Increase of PM10 over time in a 2 m$^3$ chamber from background leakage for a breathing and a talking experiment. (B) Typical increase of CO$_2$ over time during the same breathing and talking experiments from the subject.

The distribution of PM10 production rates, corrected by background leakage for breathing and talking, as well as the corresponding distributions of CO$_2$ production rates for all subjects are shown in Figures 2A and 2B. The displayed data is summarized in Table 1, which includes the average production rates as concentrations over time, the standard deviations and the average R$^2$ values of the linear regression models used to obtain the reported slopes. Note that these deviations do not represent an error, but the variation of PM and CO$_2$ production rates between participants. The average volume production rates have been calculated with the BLO-model of Johnson et al.$^{18}$, which assumes a bimodal distribution of particles with mode diameters of 1.6 µm and 2.5 µm, respectively for particles from speech. Breathing only produces particles in the smaller mode, which is linked to the bronchiolar fluid burst mechanism and amplifies the difference between the two activities. The exact formula for calculating volume concentrations can be found in the methods section.

For 13 out of 15 people tested, talking produced more particles than breathing, whereas one person emitted particles at the same rate for both activities and one person emitted more from breathing, resulting in an average increase of 2.98 ± 2.74 (one-σ standard deviation) in PM production rate ratios. There was not a clear pattern in the rate of CO$_2$ production as five people produced more CO$_2$ while talking, whereas one person produced equal amounts from both activities and nine people produced more CO$_2$ from breathing, resulting in an average ratio of 0.979 ± 0.185. The ratios are summarized in Figure 2C. Note that CO$_2$ production can be higher for either activity, but the standard deviation of 0.185 is low in comparison to the standard deviation of 2.74 for PM production rate ratios. Taking the definition of Asadi et al.$^3$, three subjects in this study can be described as talking superemitters, as their production rates exceed the mean by more than one standard deviation. With this definition, the same three people can be considered breathing superemitters. When taking the proposed definition of Holmgren et al.$^4$ instead, only one participant qualifies as a talking superemitter, as their production rate exceeds the mean by more than
Table 1. Average production rates of PM10 and CO$_2$ over time from 15 volunteers in a 2 m$^3$ chamber.

| Parameter            | Breathing | Talking   | Unit                  |
|----------------------|-----------|-----------|-----------------------|
| $m_{PM}$             | $3.11 \times 10^{-3}$ | $7.29 \times 10^{-3}$ | cm$^{-3}$ min$^{-1}$ |
| $\sigma (m_{PM})$    | $2.82 \times 10^{-3}$ | $8.41 \times 10^{-3}$ | cm$^{-3}$ min$^{-1}$ |
| $R^2 (m_{PM})$       | 0.627     | 0.806     |                       |
| $m_{PM,V}$           | $9.26 \times 10^{-3}$ | $11.2 \times 10^{-2}$ | µm$^3$ cm$^{-3}$ min$^{-1}$ |
| $\sigma (m_{PM,V})$  | $8.40 \times 10^{-3}$ | $12.9 \times 10^{-2}$ | µm$^3$ cm$^{-3}$ min$^{-1}$ |
| $m_{CO_2}$           | 143       | 139       | ppm min$^{-1}$        |
| $\sigma (m_{CO_2})$  | 29.4      | 33.2      | ppm min$^{-1}$        |
| $R^2 (m_{CO_2})$     | 0.998     | 0.998     |                       |

two standard deviations and no participant would be a breathing superemitter.

Figure 2. Boxplots of production rates from 15 participants. The median and mean are represented by a white line and a white cross, respectively, while the lower and upper box limits correspond to the lower and upper quartiles. Outliers are shown for data points exceeding the upper quartile plus 1.5 times the interquartile range (IQR) and data points lower than the lower quartile minus 1.5 times the IQR. (A) Increase of PM10 over time from respective breathing and talking experiments of 15 participants in a 2 m$^3$ chamber. (B) CO$_2$ production rates of 15 participants during the respective experiments. (C) Ratios of production rates from talking compared to breathing for each subject. Notice the break on the y scale, which was used to include an outlier.

Correlation of PM10 and CO$_2$

Figure 3A shows the increase of PM10 in relation to metabolically produced CO$_2$ for the breathing and talking experiments of the same representative subject of Figure 1. The distribution of production rates for all participants, mathematically determined with Equation 1, is shown in Figure 3B. The corresponding average production rates and their standard deviations are shown in Table 2. These deviations, again, do not represent errors, but the different production rates among volunteers. The table also includes volume production rates with CO$_2$ dependency, which have been determined by correlating the volume production rates of Table 1 with the CO$_2$ production rates. They can be used to estimate the amount of suspended SARS-CoV-2 virions, by multiplying the exhaled volume with the viral RNA concentration in oral fluid. With an average viral load of $7 \times 10^6$ ml$^{-1}$ and a maximum viral load of $2.35 \times 10^9$ ml$^{-1}$, the concentrations strongly vary between individuals and depend on the progression of the infection. The resulting, respective, average and maximum amounts of 16 and 5300 theoretically released RNA copies after 10 min of talking correlate with an approximate CO$_2$ concentration of 1.800 ppm (1.390 ppm above the background). Less copies can be expected from breathing, which, after 10 min, is estimated to result in 1 RNA copy on average or a maximum of 440 RNA copies, correlating to 1.840 ppm (1.430 ppm above the background). The above calculated values have been determined for our 2 m$^3$ chamber with homogenized air, but can be theoretically scaled to any room size, assuming the air is well mixed. The theoretically produced number of RNA copies per cubic metre per ppm of CO$_2$ $m_{RNA}$ are summarized in Table 2.
Figure 3. Correlation of PM10 and CO₂. (A) Increase of PM10 over CO₂ from talking and breathing in a 2 m³ chamber for the same representative subject from Figure 1. Distribution of (B) PM10 production rates in relation to metabolic CO₂ in a 2 m³ chamber for 15 participants. The median and mean are represented by a white line and a white cross, respectively, while the lower and upper box limits correspond to the lower and upper quartiles. Outliers are shown for data points exceeding the upper quartile plus 1.5 times the interquartile range (IQR) and data points lower than the lower quartile minus 1.5 times the IQR.

Table 2. Average increase of PM10 over CO₂ from 15 volunteers in a 2 m³ chamber.

| Parameter BREATHING | talking | Unit |
|---------------------|---------|------|
| mPMCO₂<br>σ (mPMCO₂) | 2.30 × 10⁻⁵<br>2.12 × 10⁻⁵ | cm⁻³ ppm⁻¹<br>cm⁻³ ppm⁻¹ |
| mPMCO₂,V<br>σ (mPMCO₂,V) | 6.85 × 10⁻⁵<br>6.31 × 10⁻⁵ | µm³ cm⁻³ ppm⁻¹<br>µm³ cm⁻³ ppm⁻¹ |
| mRNA<br>mRNA,max | 4.8 × 10⁻⁴<br>0.16 | m⁻³ ppm⁻¹<br|m⁻³ ppm⁻¹ |

Discussion

The presented results show that in a closed space both respiratory aerosols and metabolic CO₂ increase linearly over time and that on average talking creates more particles than breathing. As both concentrations correlate linearly with time, they correlate linearly with each other and CO₂ can be used as a proxy for airborne exhaled particles.

The results of this study can be compared to the results from other recent studies looking at respiratory aerosols. The higher particle concentrations from talking, compared to breathing observed in this study were expected, as previous studies reported the same hierarchy. It was also expected to see large variations between subjects, as standard deviations twice as high as the average production rates and outliers further than three standard deviations from the mean have been reported in other studies. These outliers have been termed superemitters or superspreaders and they might be the cause of infection in superspreading events, which have been reported in several studies.

Two of the most thorough studies investigating the particle concentrations from exhaled air have been carried out in the Expired Droplet Investigation System (EDIS), developed by Morawska et al. During their first study, average concentrations of 0.307 cm⁻³ during talking and 0.05 cm⁻³ during breathing were reported. Multiplying these values with the approximate exhalation rates of 13.5 l min⁻¹ and 12.0 l min⁻¹ for talking and breathing, respectively, leads to particle production rates of 4.14 × 10⁴ min⁻¹ and 6.00 × 10² min⁻¹, respectively. Our production rate during speech, 1.46 × 10⁴ min⁻¹ is slightly higher but lies well within the same order of magnitude. Our production rate from breathing differs slightly more, lying just outside one order of magnitude with 6.21 × 10³ min⁻¹. Looking at speech in the EDIS, volunteers were instructed to perform 10 s of voiced counting alternating with 10 s of breathing meaning subjects were only speaking half the time and bringing the production rates of their experiment and this study closer together. In a later study, which focused on the development of the BLO model in the EDIS, a particle concentration of 0.16 cm⁻³ was reported for speech, resulting in the slightly lower production rate of
2.16 $\times 10^3$ min$^{-1}$, which still lies within the same order of magnitude as our findings. Similar concentrations for breathing were observed by Holmgren et al.$^4$, who measured 0.06 cm$^{-3}$ for normal breathing and 5.3 cm$^{-3}$ for airway closure breathing, correlating to production rates of $7.2 \times 10^2$ min$^{-1}$ and $6.4 \times 10^4$ min$^{-1}$, respectively, whereas the former is more relevant to this study than the latter. Asadi et al.$^3$ investigated the particle emission from speech in relation to voice amplitude and found an average production rate of 240 min$^{-1}$ at 85 dB. As described by the authors, however, 80% of the particles were removed due to the sampling mechanism of the Aerodynamic Particle Sizer used for detection and further particles may have escaped through the only semi-confined sampling environment. Volume production rates and RNA production rates emphasize the difference between breathing and talking experiments, which might seem small when considering only number concentrations. These differ by only slightly more than a factor of two, while volume production rates differ more than one order of magnitude. This is due to the different modes during these activities, as described by Johnson et al.$^{18}$. While breathing only creates small particles from the bronchial fluid film burst mechanism (B mode) with a median diameter of 1.6 µm, talking also release particles from the larynx (L mode) with a median diameter of 2.5 µm. It therefore makes sense, that talking has been estimated to produce more than ten times the amount of airborne RNA copies. Notice that our estimation for maximum production assumes the maximum RNA copy concentration found by Wölfl et al.$^{12}$, but still uses the average particle production rate. If a superemitter of particles also happens to carry a high concentration of RNA copies in their oral fluid, their airborne RNA production rate can be even higher. This study shows that there is a strong argument for keeping indoor spaces well ventilated or using supplementary air cleaning technology. Typically, the anthropogenic metabolism is the only indoor source of CO$_2$ and, therefore, is a great proxy for suspended particles as well as indoor air quality in general. Ambient, indoor, particulate matter on the other hand has many sources and comes in concentrations several orders of magnitude higher than respiratory aerosols$^{23}$, making it impossible to monitor human expired aerosols directly. Even though specific concentrations of suspended RNA copies from an infected person may vary strongly between individuals due to different particle production rates and viral concentrations in oral fluid, CO$_2$ was shown to strongly correlate with the airborne PM concentration. The estimation of suspended RNA based on CO$_2$ measurements alone is not a reliable method for the assessment of an infection risk, as masks, different activities and air cleaners further complicate the correlation. However, high CO$_2$ concentrations measured with low cost sensors should always be a reason for concern and action should be taken to ventilate the room.

### Methods

#### Human Subjects

The University of Copenhagen Research Ethics Committee for Science and Health approved this study (Case 504-0250/21-5000) and all of the experiments were performed in accordance with relevant guidelines and regulations. All volunteers gave informed consent to participate in the study and their data has been collected anonymously.

#### Setup

An airtight chamber with 1 m length, 2 m width, and 1 m height was used as a sampling environment to simultaneously measure expelled particles from either breathing or talking and the CO$_2$ exhaled in the process. Inside the chamber, three fans were installed to mix the air along three Cartesian axes, facing each other to assure mixing. Next to this array, a probe for relative humidity and temperature was installed, as well as an infrared sensor (Sensirion SCD30) for the measurement of CO$_2$ concentrations. In the middle of the chamber, an Airbubbl (Airlabs, London, air cleaning device with F8 grade filters and 30 m$^3$ h$^{-1}$ clean air delivery rate) was placed for the removal of particles before experiments. The general array of used instrumentation is schematically shown in Figure 4. The inlet of an optical particle sizer (OPS 3330, TSI Incorporated, Shoreview, Minnesota) was connected with 3/16 inch inner diameter, 5/16 inch outer diameter, conductive tubing to the chamber and the sampling air was recirculated back inside after passing a HEPA in-line filter to keep the pressure in the chamber constant. With a flow rate of 11 min$^{-1}$, the cleaning effect through the instrument was negligible. A second connection port in the walls of the chamber was used to either increase the humidity or add filtered, dry air to create a slight overpressure. One of the chamber walls featured a quadratic opening with 80 cm width and height. This opening was covered with a sheet of Teflon, which had been prepared with a circular cutout for a short tube, that was fixed to the Teflon film in an airtight manner to create a connection between the inside and the outside of the chamber. From the outside, a rubber face mask (Moldex 7002M) was attached to the tube, so that an airtight connection could be established between a participant’s mouth and nose with the chamber. When no subject was wearing the mask, a tapered rubber plug was placed into the tube opening to seal the chamber. The setup was at a height that allowed participants to comfortably sit on a chair with adjustable height while wearing the mask. Inside the chamber, opposite the Teflon sheet, two printed DIN A3 pages with a text about particulate matter were attached to the wall as material to be read out loud for the experiments including talking.
Figure 4. 2 m³ chamber setup for measurement of respiratory particles and CO₂. OPS: Optical Particle Sizer, F1-F3: Mixing fans, T: Temperature probe, RH: Relative humidity probe, CO₂: Infrared CO₂ sensor, AB: Airbubbl.

Protocol
On each day of the study, one of the 16 volunteers (8 male, 8 female) participated in a breathing and a talking experiment. Before the subject arrived, a background measurement of particle leakage into the chamber was taken. The OPS was measuring with a sample length of 1 minute, while CO₂ concentrations were registered every 30 seconds. With all fans running and the tube plug removed, air from a humidifier was released into the chamber, until a relative humidity (RH) of 50 % was reached. With reinserted tube plug, the Airbubbl was then turned on to clean the air until the PM10 concentration was below 0.02 cm⁻³. The Airbubbl was then turned off and the background increase of particles was measured for at least 30 minutes. The Airbubbl was then turned on again to reduce particle concentration below 0.02 cm⁻³ after making sure, that the relative humidity remained at 50 %. Then, the Airbubbl was turned off and three to five samples of PM background concentrations were taken. During this, clean air was introduced into the chamber through a HEPA filter, to create a slight overpressure. This assured, that clean air from the chamber was exiting the chamber instead of outside air entering, when the participant removed the tube plug and put on the mask. For the first experiment, the subject was instructed to breath normally through the nose for ten minutes. Afterwards, the mask was taken off and the tube plug reinserted. After taking at least three more samples, the chamber was flushed with room air to remove the additional CO₂, rehumidified to 50 % RH and cleaned with the Airbubbl to reduce the PM concentration. The procedure of the first experiment was then repeated, but with the participant reading out loud the printed text inside the chamber instead of only breathing.

Between subjects, the chamber was sterilised using an ozone generator, which has been shown to effectively remove both viruses²⁴ and bacteria²⁵. The mask, tube and plug were thoroughly washed with water and soap and then disinfected.

Data Treatment
All data was analysed in Python. First, a linear regression model for background PM leakage into the chamber over at least 30 minutes was determined for each measurement day. Before each breathing and talking experiment, the average background concentration of particles was measured with the Airbubbl turned off immediately before establishing the connection between subject and chamber. This background concentration was then subtracted from the data of the respective experiment. Then, linear regression models for PM10 and CO₂ over time were created. The background leakage slope for PM was subtracted from the PM10 slope to yield the actual increase from the respective activity. Since two measurement instruments were used and their sampling intervals did not exactly align, the PM increase over CO₂ \( m_{PMCO₂} \) in cm⁻³ ppm⁻¹ was calculated as

\[
m_{PMCO₂} = \frac{m_{PM}}{m_{CO₂}},
\]

where \( m_{PM} \) is the background leakage corrected increase of PM over time in cm⁻³ min⁻¹ and \( m_{CO₂} \) is the increase of CO₂ over time in ppm min⁻¹. The PM concentration \( c_{PM} \) in cm⁻³ could then be calculated with

\[
c_{PM}(c_{CO₂}) = m_{PMCO₂}(c_{CO₂} - b_{CO₂}) + b_{PM},
\]
where $c_{CO_2}$ is the CO$_2$ concentration in ppm, $m_{PM.CO_2}$ is the PM increase over CO$_2$ in cm$^{-3}$ ppm$^{-1}$, calculated from equation (1). $b_{CO_2}$ is the intercept of the CO$_2$ linear regression model in ppm and $b_{PM}$ is the intercept of the PM linear regression model in cm$^{-3}$.

### Estimation of Volume Concentrations

The setup was not designed to measure volume concentrations, as particles were too diluted in the 2 m$^3$ chamber to obtain a size distribution. Instead, the size distribution described by Johnson et al.$^{18}$ was used to estimate the volume of airborne particles. Of their BLO-model, only modes B (bronchiolar fluid film burst) and L (larynx mode) were used for particles from speech, as the O (oral) mode was measured using droplet deposition analysis and these particles are not expected to be airborne. The volume distributions were estimated with

$$dV = \ln 10 \times \sum_{i=1}^{2} \frac{\pi}{6} D^3 \left( \frac{C_{n_i}}{\sqrt{2 \pi \ln GSD_i}} \right) \exp \left( -\frac{(\ln(D) - \ln CMD_i)^2}{2(\ln GSD_i)^2} \right),$$  

where $D$ is the particle diameter and $C_{n_i}$ is the number concentration. $GSD_i$ and $CMD_i$ are the geometric standard deviations and count median diameters of modes B and L and have been taken from Johnson et al.$^{18}$. For particles from breathing, only the B mode was considered. Volume concentrations were then calculated by simply integrating over the distributions.

### Data Availability

All relevant data from the study are available from the corresponding author on reasonable request.

### References

1. Morawska, L. & Milton, D. K. It Is Time to Address Airborne Transmission of Coronavirus Disease 2019 (COVID-19). *Clin. Infect. Dis.* **2019**, 1–4, DOI: 10.1093/cid/ciaa939 (2020).
2. Morawska, L. et al. Size distribution and sites of origin of droplets expelled from the human respiratory tract during expiratory activities. *J. Aerosol Sci.* **40**, 256–269, DOI: 10.1016/j.jaerosci.2008.11.002 (2009).
3. Asadi, S. et al. Aerosol emission and superemission during human speech increase with voice loudness. *Sci. Reports* **9**, 1–10, DOI: 10.1038/s41598-019-38808-z (2019).
4. Holmgren, H., Ljungström, E., Almstrand, A. C., Bake, B. & Olin, A. C. Size distribution of exhaled particles in the range from 0.01 to 2.0µm. *J. Aerosol Sci.* **41**, 439–446, DOI: 10.1016/j.jaerosci.2010.02.011 (2010).
5. Xie, X., Li, Y., Chwang, A. T., Ho, P. L. & Seto, W. H. How far droplets can move in indoor environments - revisiting the Wells evaporation-falling curve. *Indoor Air* **17**, 211–225, DOI: 10.1111/j.1600-0668.2007.00469.x (2007).
6. Santarpia, J. L. et al. Aerosol and surface contamination of SARS-CoV-2 observed in quarantine and isolation care. *Sci. Reports* **10**, 1–8, DOI: 10.1038/s41598-020-69286-3 (2020).
7. Liu, Y. et al. Aerodynamic analysis of SARS-CoV-2 in two Wuhan hospitals. *Nature* **582**, 557–560, DOI: 10.1038/s41586-020-2271-3 (2020).
8. van Doremalen, N. et al. Aerosol and Surface Stability of SARS-CoV-2 as Compared with SARS-CoV-1. *The New Engl. J. Medicine* **382**, 1564–1567, DOI: 10.1056/NEJMc2004973 (2020).
9. Yu, I. T. et al. Evidence of Airborne Transmission of the Severe Acute Respiratory Syndrome Virus. *New Engl. J. Medicine* **350**, 1731–1739, DOI: 10.1056/nejmoa032867 (2004).
10. Kim, S. H. et al. Extensive Viable Middle East Respiratory Syndrome (MERS) Coronavirus Contamination in Air and Surrounding Environment in MERS Isolation Wards. *Clin. Infect. Dis.* **63**, 363–369, DOI: 10.1093/cid/ciw239 (2016).
11. Yan, J. et al. Infectious virus in exhaled breath of symptomatic seasonal influenza cases from a college community. *Proc. Natl. Acad. Sci. United States Am.* **115**, 1081–1086, DOI: 10.1073/pnas.1716561115 (2018).
12. Wölfel, R. et al. Virological assessment of hospitalized patients with COVID-2019. *Nature* **581**, 465–469, DOI: 10.1038/s41586-020-2196-x (2020).
13. Du, C. R. et al. Effect of ventilation improvement during a tuberculosis outbreak in underventilated university buildings. *Indoor Air* **30**, 422–432, DOI: 10.1111/ina.12639 (2020).
14. Rudnick, S. N. & Milton, D. K. Risk of indoor airborne infection transmission estimated from carbon dioxide concentration. *Indoor Air* **13**, 237–245, DOI: 10.1034/j.1600-0668.2003.00189.x (2003).
15. Peng, Z. & Jimenez, J. L. Exhaled CO2 as COVID-19 infection risk proxy for different indoor environments and activities. medRxiv 2020.09.09.20191676 (2020).
16. Harrichandra, A., Ierardi, A. M. & Pavilonis, B. An estimation of airborne SARS-CoV-2 infection transmission risk in New York City nail salons. Toxicol. Ind. Heal. 36, 634–643, DOI: 10.1177/0748233720964650 (2020).
17. Burrdige, H. C., Fan, S., Jones, R. L., Noakes, C. J. & Linden, P. Predictive and retrospective modelling of airborne infection risk using monitored carbon dioxide. arXiv (2021).
18. Johnson, G. R. et al. Modality of human expired aerosol size distributions. J. Aerosol Sci. 42, 839–851, DOI: 10.1016/j.jaerosci.2011.07.009 (2011).
19. Edwards, D. A. et al. Inhaling to mitigate exhaled bioaerosols. Proc. Natl. Acad. Sci. United States Am. 101, 17383–17388, DOI: 10.1073/pnas.0408159101 (2004).
20. Lloyd-Smith, J. O., Schreiber, S. J., Kopp, P. E. & Getz, W. M. Superspreading and the effect of individual variation on disease emergence. Nature 438, 355–359, DOI: 10.1038/nature04153 (2005).
21. Miller, S. L. et al. Transmission of SARS-CoV-2 by inhalation of respiratory aerosol in the Skagit Valley Chorale superspreading event. Indoor Air 1–17, DOI: 10.1111/ina.12751 (2020).
22. Gupta, J. K., Lin, C. H. & Chen, Q. Characterizing exhaled airflow from breathing and talking. Indoor Air 20, 31–39, DOI: 10.1111/j.1600-0668.2009.00623.x (2010).
23. Patel, S. et al. Indoor Particulate Matter during HOMEChem: Concentrations, Size Distributions, and Exposures. Environ. Sci. Technol. 54, 7107–7116, DOI: 10.1021/acs.est.0c00740 (2020).
24. Hudson, J. B., Sharma, M. & Petric, M. Inactivation of Norovirus by ozone gas in conditions relevant to healthcare. J. Hosp. Infect. 66, 40–45, DOI: 10.1016/j.jhin.2006.12.021 (2007).
25. Sharma, M. & Hudson, J. B. Ozone gas is an effective and practical antibacterial agent. Am. J. Infect. Control. 36, 559–563, DOI: 10.1016/j.ajic.2007.10.021 (2008).

Author contributions statement
N.K., H.S.R., S.K. and M.S.J. designed research, N.K. carried out experiments and analyzed the data, N.K. and A.A. wrote the manuscript, N.K. created Figure 4 and all authors reviewed the manuscript.

Additional information
Competing interests: The authors declare no competing interests.