Distinct regimes of particle and virus abundance explain face mask efficacy for COVID-19

Authors: Yafang Cheng\(^{1,\ast, **} \), Nan Ma\(^ {2*} \), Christian Witt\(^ {3} \), Steffen Rapp\(^ {4} \), Philipp Wild\(^ {4} \), Meinrat O. Andreae\(^ {1,5,6} \), Ulrich Pöschl\(^ {1} \), Hang Su\(^ {7,1} \)*.

Affiliations:

1 Max Planck Institute for Chemistry, Mainz 55128, Germany.
2 Institute for Environmental and Climate Research, Jinan University, Guangzhou 511443, China.
3 Department of Outpatient Pneumology, Charité Universitätsmedizin Berlin, Campus Charité Mitte, Charitéplatz 1, 10117, Berlin, Germany.
4 University Medical Center of the Johannes Gutenberg-University Mainz, Germany.
5 Scripps Institution of Oceanography, University of California San Diego, La Jolla, CA 92093, USA.
6 Department of Geology and Geophysics, King Saud University, P.O Box. 2455, 11451 Riyadh, Saudi Arabia.
7 State Environmental Protection Key Laboratory of Formation and Prevention of Urban Air Pollution Complex, Shanghai Academy of Environmental Sciences, Shanghai, China.

* These authors contributed equally to this work.

† Correspondence to: H.S. (h.su@mpic.de) and Y.C. (yafang.cheng@mpic.de)

Abstract:
Airborne transmission is an important transmission pathway for viruses, including SARS-CoV-2. Regions with a higher proportion of people wearing masks show better control of COVID-19, but the effectiveness of masks is still under debate due to their limited and variable efficiencies in removing respiratory particles. Here, we analyze experimental data and perform model calculations to show that this contrast can be explained by the different abundance regimes between particles and viruses. Upon short-term exposure, respiratory particles are usually in a particle-rich regime, but respiratory viruses are often in a virus-limited regime where the numbers of viruses inhaled by susceptible people are below or close to the infectious dose. This virus-limited regime ensures mask efficacy and synergy of multiple preventive measures in reducing the infection risk.
Main Text:

Airborne transmission is regarded as one of the main pathways for the transmission of viruses that lead to infectious respiratory deceases, including the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) (1), and wearing masks has been widely advocated to minimize transmission and protect people. Though commonly used, the effectiveness of surgical masks is still under debate. Compared to N95 respirators, surgical masks show a higher and more variable penetration rate (e.g., from ~30% to 70%) (2, 3), and are often considered insufficient to protect people. However, observational data show that regions with a higher percentage of the population wearing masks have better control of coronavirus disease 2019 (COVID-19) (4-6). So how to explain the apparently conflicting results that masks with relatively high penetration rates may still have a significant impact on airborne virus transmission and the spread of COVID-19?

Here, we combine knowledge of aerosol science and medical research with recent progress and literature data to explain the reason behind this contrast, which provides a basis for obtaining quantitative estimates for the effectiveness of wearing face masks.

For a given time period, the probability of inhaling more than certain amount of viruses (e.g., critical infectious dose), $P$, is a function of the ambient concentration of airborne viruses $C$. Figure 1A shows the calculated probability of inhaling more than or equal to one virus as a function of $C$ from a series of Poisson cumulative probability functions. When $C$ is extremely high (virus-rich regime, Fig. 1B), the value of $P$ is 1 and is not sensitive to $C$. In this case, wearing masks may have limited effects in reducing the inhalation probability. However, in the virus-limited regime where $P$ varies between 0 and 1, the change of $C$ will also lead to a change in $P$. In this case, wearing masks can influence the inhalation probability and thus becomes effective (Fig. 1C).

Respiratory particles, as carriers of respiratory viruses, are often used to visualize and represent the transmission of airborne viruses. We first look at the abundance regimes of respiratory particles. Figure 2 shows the size distributions of particles emitted by different activities (7-9). Taking a representative average of activities given in (10), we find that people can emit a total number of about $3 \times 10^6$ particles in a 30 min sampling period (Sect S1). This extremely large number shows that we are always in a respiratory particle-rich regime. Even after wearing surgical masks, the low collection efficiency still leaves over millions of particles emitted, maintaining a particle-rich regime (green dots in Figs. 1B and 1C). In other words, the human-emitted particle concentration is so high that we cannot avoid inhaling particles generated by another person even when wearing a mask. But does a respiratory particle-rich regime imply a respiratory virus-rich regime?

For exhaled respiratory viruses, as we are not aware of any direct measurement of SARS-CoV-2 emissions, we analyze the recent results for multiple other viruses during a 30-min collection in Leung et al. (2020) (10). This study has a relatively large sample number (246 samples) and diverse virus types (coronaviruses, influenza viruses and rhinoviruses). Moreover, the samples have been collected for both particles above and below ~5 µm, and individual contributions from aerosol mode (< 5 µm) and droplet mode (> 5 µm) particles can be separated. As many samples in Leung et al. (2020) (10) return a virus load signal below the detection limit, we reconstructed the mathematical expectation based on the percentage of positive cases and standard deviation of virus distributions (detailed in Sect S2).
Fig. 1. Schematic illustration of abundance regimes of airborne transmission. (A) The black dashed line represents the critical concentration $C$ for people to inhale a threshold number of viruses. We can see a “virus-rich” regime or a “virus-limited” regime for cases when inhaled virus concentration is above or below the threshold, respectively. Under a particle/virus-rich regime, people will always inhale large numbers of particles/viruses and wearing a mask has a limited effect if the resultant concentration is still above the threshold. Under a virus-limited regime, wearing a mask will further reduce the virus concentration and the risk to be infected. The red/green dashed lines represent exemplary virus/particle concentrations $C$ corresponding to the virus-limited and virus-rich regimes, respectively. (B) virus-rich and particle-rich regime; (C) virus-limited and particle-rich regime.
Fig. 2. **Particle size distribution from different human activities.** Volume size distribution of released particles by sneezing (A), coughing (B), speaking (C) and breathing (D) and those with surgical and N95 masks. Circles represent measurements and solid lines show bimodal fits to measurements. Here, the distribution of exhaled particles for each human activity is also plotted for reference. $V$ and $D_p$ represents the particle volume and diameter, respectively.

It turns out that most of the time only a few exhaled respiratory particles contain viruses. In contrast to the high concentration of respiratory particles, the emitted virus concentration is very low with median $N_{\text{sample}}$ (number of exhaled viruses in a 30-min sample) of $\sim 0.96$ for coronaviruses (HCoV-NL63, -OC43, -229E and -HKU1), $\sim 0.64$ for influenza viruses (A and B) and $\sim 4.9$ for rhinoviruses. These low $N_{\text{sample}}$ values all fall into a virus-limited regime and are even lower than the reported critical infectious dose (e.g., a few tens to thousands viruses) for several viruses (11-14). For the coronavirus SARS-CoV-2, we examined its virus regime based on $N_{30}$ (virus number per 30-min inhalation of $\sim 240$ L indoor air) in hospitals and health centers, where high concentrations of SARS-CoV-2 are expected. In Fangcang Hospital in Wuhan, the measured airborne SARS-CoV-2 concentrations varied from undetected to 0.019 L$^{-1}$ air (15), corresponding to $\sim 0$ to 4 viruses per 30-min inhalation. Given a reported infectious dose (hamsters) of $< 1000$ viruses for SARS-CoV-2 (14), a small $N_{30}$ of $\sim 0$ to 4 viruses is likely in a virus-limited regime. Similar virus regimes are also found in other studies, e.g., $N_{30}$ of SARS-CoV-2 in U.S. and Singapore medical centers/hospitals have been found to vary from undetected to $\sim 209$-2086 (Fig. 3A) (16-18), which are either within or overlapped with the virus-limited regime.
**Fig 3. Abundance regime of airborne transmission of viruses.** (A) The solid lines show the infection probability ($P_{\text{infect}}$) after inhaling certain number of viruses at different critical infectious doses. Colored dots show the 30-min inhaled virus numbers ($N_{30}$) calculated based on reported airborne SARS-CoV-2 concentrations in medical centers/hospitals. Open circles show the 30-min exhaled number of coronavirus, influenza virus and rhinovirus. Shading areas show the standard deviation. (B) The same as (A) except for the calculation of the solid lines. The solid lines have considered the individual differences in a large population, in which $N_{30,\text{med}}$ represents the median 30-min inhaled virus number of the whole population. The inhaled virus number is assumed to follow a lognormal distribution with a $\sigma$ of ~ 2 (Sect S2). The virus-rich and virus-limited regime is defined as $N_{30} (P_{\text{infect}} > 0.95)$ and $N_{30} (P_{\text{infect}} \leq 0.95)$, respectively.

To link the results of exhalation samples with ambient samples, we design a scenario with patient density, space areas, and ventilation conditions emulating Fangcang Hospital in Wuhan (Sect S4). Under this scenario, we can calculate the ambient concentrations for a given virus emission rate. Based on the emission rates of Leung et al. (2020), we calculated an $N_{30}$ of undetectable to ~ 52 (5% to 95%) for coronavirus, influenza and rhinovirus in the Fangcang Hospital. Because this concentration range of other viruses overlapped with that of the observed airborne SARS-CoV-2 ($N_{30} \sim 0$ to 4) in the Fangcang Hospital (15), we expect that the exhaled samples of SARS-CoV-2 may show a similar virus regime as those of other viruses.

Individual differences, different development stage of symptoms (21), ventilation, and heterogeneity of virus distribution in indoor environments will cause variability of virus number in the exhaled and inhaled samples. For example in Leung et al. (2020) (10), despite of a low median $N_{\text{sample}}$ values of ~0.6 to 5, the maximum $N_{\text{sample}}$ values reached ~ $4 \times 10^3$ to $4 \times 10^5$ viruses. The degree of this variability is a key parameter in the assessment of infectious risks, selection of protection devices/strategies and uncertainty analysis of these assessments. Based on measurement data in sputum samples (21), we find that the number of SARS-CoV-2 viruses in
respiratory liquid shows a large variability, and follows a lognormal distribution with a $\sigma$ of $\sim 2$ (Sect S2). As shown Fig. 3B, such variability changed the shape of infection probability ($P_{\text{infect}}$) curve and expanded the range of virus-limited regimes where wearing masks are effective. Besides, the large variability also suggests that the limited sample numbers and virus measurements commonly used may introducing uncertainties to the assessment (Sect S5), which may explain why early studies that have investigated whether masks reduce infection in randomized controlled trials obtained results that were partly inconsistent (22-25).

**Fig. 4. Reduced chance of COVID-19 transmission with masks.** The curves represent the percentage change of $P_{\text{infect}}$ caused by mask use due to the change of $N_{30}$. The blue and red lines represent the results with surgical (blue lines) and N95 masks (red lines) while the solid and dashed lines represent the results for a critical dose of 1 virus and 10000 viruses, respectively. The dependence of $P_{\text{infect}}$ on $N_{30}$ used here was assumed the same as in Fig. 3B.

Figure 4 shows the reduced chance of COVID-19 transmission with surgical and N95 masks calculated from Fig. 3B, i.e., the percentage change of $P_{\text{infect}}$ caused by mask use due to the change of $N_{30}$. It is commonly assumed that the percentage change of $P_{\text{infect}}$ is proportional to the percentage change of $N_{30}$. In this way, wearing the same mask will have the same impact on the virus transmission at any $P_{\text{infect}}$. However, our analysis shows a nonlinear effect of mask uses on the virus transmission, which strongly depends on the present infection probability, $P_{\text{infect}}$, or $N_{30}$. As shown in Fig. 4, at high $P_{\text{infect}}$, wearing masks have a minor effect on $P_{\text{infect}}$ while at low $P_{\text{infect}}$, wearing masks become very efficient. According to the ratio of the reproduction rate ($\sim 2$-$7$) for SARS-CoV-2 to the average daily contact number ($\sim 10$-$25$) (26, 27), we can estimate an upper limit of the effective $P_{\text{infect}}$ of $\sim 10\%$ to $70\%$ for airborne transmission in large populations, suggesting the ubiquity of a virus-limited regime for SARS-CoV-2. As shown in Fig. 4, in this range of $P_{\text{infect}}$, wearing masks (both surgical and N95 masks) may largely reduce the chance of COVID-19 transmission. This is consistent with the results of 172 observational studies across
16 countries and six continents which have shown a large reduction in risk of infection by face mask use (5). More importantly, the increasing effectiveness of mask use at lower \( P_{\text{infect}} \) and \( N_{\text{30}} \) suggests synergistic effects of multiple preventive measures in reducing the infection risk.

Concerning the relative importance of aerosol mode vs droplet mode, we find that the aerosol mode, despite of much smaller particle volumes, show a virus number similar to or even slightly higher than that of the droplet mode for both ambient and exhaled samples: \( N_{\text{30}} \) (aerosol mode vs droplet mode) of \(~5.1 \) vs \(~1.4\) for SARS-CoV-2 in the Fangcang Hospital (Table S7); and \( N_{\text{sample}} \) (aerosol mode vs droplet mode) of \(0.75\) vs \(0.21\) for coronaviruses (HCoV-NL63, -OC43, -229E and -HKU1), of \(0.55\) vs \(0.091\) for influenza viruses (A and B) and of \(4.7\) vs \(0.18\) for rhinoviruses, respectively. This suggests a much higher virus concentration per particle volume in the aerosol mode than that in the droplet mode. Because the amount of bioaerosols or compounds delivered in particles is proportional to its concentration in the bulk fluid used to generate the particles, and is independent of the investigated particulate type (19). If the aerosol and droplet modes are mainly generated from the lower and upper respiratory tracts respectively (20), the higher concentration of viruses in the lung fluid (i.e., sputum samples) show much higher virus concentrations than throat and nasal swabs (21) may explain the high virus concentration in the aerosol mode.

The abundance regimes, size dependence, and individual differences have important implications in epidemic prevention. The large fraction of virus in the aerosol mode suggests a higher risk than expected, because small particles have a longer lifetime in the air and thus can accumulate to a threshold infection level. This also shows that the greatest danger is in spaces with large number of people and poor ventilation, where virus accumulates in the air over long times. Long period of release, long residence time, and long period of exposure combine to maximize risks. Besides, aerosol mode particles also have a higher penetration rate, and probability to reach the lower respiratory tract (e.g., lung) (29, 30), we thus expect that the aerosol mode can cause more severe infectious symptoms than the droplet mode particles in view of the infection mechanism/nature of SARS-CoV-2.

However, our results show that the airborne transmission of SARS-CoV-2 is most likely in a virus-limited regime. In this regime, any preventive measure (such as wearing masks, ventilation, social distancing) that reduces the inhaled particles concentrations will reduce the infection probability. The increasing efficiency of preventive measures at lower virus concentration also suggests that the more measures used, the more effective each measure will be in containing the virus transmission. For example, when both sources (infector) and susceptible people were wearing masks, the inhaled virus concentration will be further reduced, thereby further improving the efficacy of the mask and forming positive feedback. Besides, because the inhaled dose also affects the severity of the infection (14), masks can still be useful even if the reduced dose still leads to an infection. The differences between abundance regimes are not limited to respiratory particles and viruses, but may also exist between different types of viruses. Viruses of higher emission/exhalation rates, longer lifetime and lower infectious dose may result in a virus-rich regime and thus a high basic reproduction number (most likely in the case of measles (28)).

The orders-of-magnitude differences in emitted virus concentrations between individuals suggest that some patients can emit far more viruses and become super spreaders. According to Wölfel et al. (2020) (21), pharyngeal virus shedding was very high during the first week of symptoms. The
large variability also suggests that even if the median value is in a virus-limited regime, an individual patient, i.e., a super spreader, may still create a virus-rich regime, where wearing surgical masks would provide insufficient protections. To better deal with such cases, stricter measures, including wearing N95 masks become critical in preventing virus transmission. This is also supported by the fact that wearing N95 masks (and eye protection) leads to a low rate of infection despite of close contact with infectious people (among the 40000 medical staffs, almost no one got infected when wearing N95 masks in Wuhan, [link](http://www.ccdi.gov.cn/yaowen/202003/t20200323_214056.html)).

### References and Notes:

1. L. Morawska, D. K. Milton, It is Time to Address Airborne Transmission of COVID-19. *Clinical Infectious Diseases*, (2020).
2. S. A. Grinshpun *et al.*, Performance of an N95 Filtering Facepiece Particulate Respirator and a Surgical Mask During Human Breathing: Two Pathways for Particle Penetration. *Journal of Occupational and Environmental Hygiene* **6**, 593-603 (2009).
3. T. Oberg, L. M. Brosseau, Surgical mask filter and fit performance. *American Journal of Infection Control* **36**, 276-282 (2008).
4. T.-C. Hsiao, H.-C. Chuang, S. M. Griffith, S.-J. Chen, L.-H. Young, COVID-19: An Aerosol’s Point of View from Expiration to Transmission to Viral-mechanism. *Aerosol and Air Quality Research*, 905-910 (2020).
5. D. K. Chu *et al.*, Physical distancing, face masks, and eye protection to prevent person-to-person transmission of SARS-CoV-2 and COVID-19: a systematic review and meta-analysis. *The Lancet* **395**, 1973-1987 (2020).
6. R. Zhang, Y. Li, A. L. Zhang, Y. Wang, M. J. Molina, Identifying airborne transmission as the dominant route for the spread of COVID-19. *Proc. Natl. Acad. Sci.* **117**, 14857-14863 (2020).
7. C. Y. H. Chao *et al.*, Characterization of expiration air jets and droplet size distributions immediately at the mouth opening. *Journal of aerosol science* **40**, 122-133 (2009).
8. J. P. Duguid, The size and the duration of air-carriage of respiratory droplets and droplet-nuclei. *The Journal of hygiene* **44**, 471-479 (1946).
9. H. Holmgren, E. Ljungström, A.-C. Almstrand, B. Bake, A.-C. Olin, Size distribution of exhaled particles in the range from 0.01 to 2.0μm. *Journal of Aerosol Science* **41**, 439-446 (2010).
10. N. H. L. Leung *et al.*, Respiratory virus shedding in exhaled breath and efficacy of face masks. *Nature Medicine* **26**, 676-680 (2020).
11. K. A. Callow, H. F. Parry, M. Sergeant, D. A. J. Tyrrell, The time course of the immune response to experimental coronavirus infection of man. *Epidemiology and Infection* **105**, 435-446 (1990).
12. T. Watanabe, T. A. Bartrand, M. H. Weir, T. Omura, C. N. Haas, Development of a Dose-Response Model for SARS Coronavirus. *Risk Analysis* **30**, 1129-1138 (2010).
13. A. Roberts *et al.*, Severe Acute Respiratory Syndrome Coronavirus Infection of Golden Syrian Hamsters. *Journal of Virology* **79**, 503-511 (2005).
14. M. Imai *et al.*, Syrian hamsters as a small animal model for SARS-CoV-2 infection and countermeasure development. *Proc. Natl. Acad. Sci.* **117**, 16587-16595 (2020).
15. Y. Liu et al., Aerodynamic analysis of SARS-CoV-2 in two Wuhan hospitals. Nature 582, 557-560 (2020).
16. P. Y. Chia et al., Detection of air and surface contamination by SARS-CoV-2 in hospital rooms of infected patients. Nat. Commun. 11, 2800 (2020).
17. J. L. Santarpia et al., Aerosol and Surface Transmission Potential of SARS-CoV-2. medRxiv, 2020.03.23.20039446 (2020).
18. J. A. Lednicky et al., Collection of SARS-CoV-2 Virus from the Air of a Clinic within a University Student Health Care Center and Analyses of the Viral Genomic Sequence. Aerosol and Air Quality Research 20, 1167-1171 (2020).
19. M. O. Fernandez et al., Assessing the airborne survival of bacteria in populations of aerosol droplets with a novel technology. Journal of The Royal Society Interface 16, 20180779 (2019).
20. B. Bake, P. Larsson, G. Ljungkvist, E. Ljungström, A. C. Olin, Exhaled particles and small airways. Respiratory Research 20, 8 (2019).
21. R. Wölfel et al., Virological assessment of hospitalized patients with COVID-19. Nature 581, 465-469 (2020).
22. C. R. MacIntyre et al., Efficacy of face masks and respirators in preventing upper respiratory tract bacterial colonization and co-infection in hospital healthcare workers. Preventive medicine 62, 1-7 (2014).
23. C. R. MacIntyre et al., A randomized clinical trial of three options for N95 respirators and medical masks in health workers. American journal of respiratory and critical care medicine 187, 960-966 (2013).
24. M. Loeb et al., Surgical mask vs N95 respirator for preventing influenza among healthcare workers: a randomized trial. Jama 302, 1865-1871 (2009).
25. L. J. Radonovich, Jr et al., N95 Respirators vs Medical Masks for Preventing Influenza Among Health Care Personnel: A Randomized Clinical Trial. Jama 322, 824-833 (2019).
26. J. Zhang et al., Changes in contact patterns shape the dynamics of the COVID-19 outbreak in China. Science 368, 1481-1486 (2020).
27. S. Y. Del Valle, J. M. Hyman, H. W. Hethcote, S. G. Eubank, Mixing patterns between age groups in social networks. Social Networks 29, 539-554 (2007).
28. F. M. Guerra et al., The basic reproduction number R₀ of measles: a systematic review. The Lancet Infectious Diseases 17, e420-e428 (2017).
29. W. G. Kreyling, M. Semmler-Behnke, W. Möller, Health implications of nanoparticles. Journal of Nanoparticle Research 8, 543-562 (2006).
30. W. G. Kreyling, M. Semmler-Behnke, W. Möller, Ultrafine particle-lung interactions: does size matter? Journal of aerosol medicine : the official journal of the International Society for Aerosols in Medicine 19, 74-83 (2006).
31. S. A. Grinshpun et al., Performance of an N95 filtering facepiece particulate respirator and a surgical mask during human breathing: two pathways for particle penetration. Journal of occupational and environmental hygiene 6, 593-603 (2009).
32. A. Weber et al., Aerosol penetration and leakae characteristics of masks used in the health care industry. American Journal of Infection Control 21, 167-173 (1993).
33. J. Zhao et al., Particle Mass Concentrations and Number Size Distributions in 40 Homes in Germany: Indoor-to-outdoor Relationships, Diurnal and Seasonal Variation. Aerosol and Air Quality Research 20, 576-589 (2020).
34. M. Nicas, W. W. Nazaroff, A. Hubbard, Toward Understanding the Risk of Secondary Airborne Infection: Emission of Respirable Pathogens. *Journal of Occupational and Environmental Hygiene* 2, 143-154 (2005).
35. N. van Doremalen *et al.*, Aerosol and Surface Stability of SARS-CoV-2 as Compared with SARS-CoV-1. *New England Journal of Medicine* 382, 1564-1567 (2020).
36. S. L. Miller *et al.*, Transmission of SARS-CoV-2 by inhalation of respiratory aerosol in the Skagit Valley Chorale superspreading event. *MedRxiv*, (2020).
37. T. L. Thatcher, A. C. K. Lai, R. Moreno-Jackson, R. G. Sextro, W. W. Nazaroff, Effects of room furnishings and air speed on particle deposition rates indoors. *Atmospheric Environment* 36, 1811-1819 (2002).
38. Y. Drossinos, N. I. Stilianakis, What aerosol physics tells us about airborne pathogen transmission. *Aerosol Science and Technology* 54, 639-643 (2020).
39. G. Chowell, N. W. Hengartner, C. Castillo-Chavez, P. W. Fenimore, J. M. Hyman, The basic reproductive number of Ebola and the effects of public health measures: the cases of Congo and Uganda. *Journal of Theoretical Biology* 229, 119-126 (2004).
40. J. Zhang *et al.*, Changes in contact patterns shape the dynamics of the COVID-19 outbreak in China. *Science* 368, 1481-1486 (2020).
41. H. Wang *et al.*, Phase-adjusted estimation of the number of Coronavirus Disease 2019 cases in Wuhan, China. *Cell Discovery* 6, 1-8 (2020).

**Acknowledgments:** This study is supported by the Max Planck Society (MPG); **Funding:** Y.C. thanks the Minerva Program of MPG; **Author contributions:** Y.C. and H.S. design and led the study. H.S., Y.C. and N.M. perform the research. U.P. and M.O.A. discussed the results. C.W., S.R. and P.W. commented on the manuscript. H.S. and Y.C. wrote the manuscript with inputs from N.M. and all coauthors; **Competing interests:** Authors declare no competing interests; and **Data and materials availability:** All data is available in the main text or the supplementary materials.

**Supplementary Materials:**
Supplementary Text S1 to S6
Figs. S1 to S7
Tables S1 to S7